5. REVISIONS TO THE OU 4-13 REMEDIAL INVESTIGATION FEASIBILITY STUDY WORK PLAN OU 4-13 FIELD SAMPLING

This section discusses documented revisions to the field sampling plan and the RI/FS Work Plan (McCormick et al. 1997). These revisions were documented, reviewed, and approved using LMITCO Document Action Requests (DARs).

5.1 CFA-04 Revisions to the Field Sampling Plan

Several areas were identified during the field investigation at the CFA-04 site that required additional samples to be collected and/or a modification of the existing sampling design. Changes were made per the DARs discussed below.

Additional samples were collected in the CFA-04 Pond (ER-DAR-681). Low areas in the pond bottom were identified as a result of the topographic survey where liquids containing mercury and other contaminants may have concentrated. Additional sample locations were biased to these areas of the pond. The samples were collected to determine the maximum concentrations of potential contaminants in the surface sediments of the pond. They were analyzed for mercury, zirconium, and arsenic.

Samples were collected (ER-DAR-681) in the staging area immediately north of the pond. Mercury retort soil treatment equipment was located in the staging area during the time-critical removal action in 1995. Surface samples were collected in a random distribution to determine the maximum concentrations of mercury, zirconium, and arsenic.

Samples were collected (ER-DAR-681) from tanks used during the mercury retort process. The tanks contained water from decontamination operations. The analyses were used to determine the waste disposal options for the tanks and contents.

The conditions for collection of samples for gamma screen analyses were revised (ER-DAR-684). Prior to approval of this DAR, samples for gamma screen analysis were collected and analyzed to ensure compliance with shipping regulations. This change, implemented as a cost saving measure, allowed the radiation control technician to use site process knowledge to eliminate the gamma screen prior to shipping.

Additional samples were collected from the trenches in the Western Anomaly near the CFA-04 Pond (ER-DAR-790). These samples replaced the original samples collected per the OU 4-13 Field Sampling Plan, because the holding times for these samples were not attained by the laboratory.

The depth of sediments in the pond was measured (ER-DAR-795). These measurements will be used in the feasibility study to determine the amount of soil in the pond bottom.

Additional samples were collected from the pond bottom and windblown areas in July 1998. These data are used in the risk assessment and to determine the waste status of the soil for the FS.

5.2 CFA-08 Revisions to the Field Sampling Plan

Twenty subsurface samples were added to the CFA-08 drainfield sampling design (ER-DAR-681). Samples were collected to a depth of 2.1 m (7 ft.) along the three pipelines between the CFA-657 Pumphouse and the drainfield. These samples were collected to determine if contaminated soil was

present in the vicinity of the drainfield delivery pipelines, from leakage. Samples were collected in the vicinity of the pipelines and analyzed for Sr-90, which was used as an indicator contaminant. If Sr-90 was detected in a subsurface soil sample additional samples were analyzed for other potential contaminants.

Further evaluation of the pipelines was proposed using a camera system (ER-DAR-790). The pipes were excavated at a location approximately half way between the CFA-657 Pumphouse and the drainfield and holes were drilled into each pipe. Water and sludge was present in the pipes, consequently, the camera system could not be used and additional samples were collected (ER-DAR-847). Samples were collected from the water and sludge in the pipelines and analyzed to determine their toxicity characteristics. These analyses will be used to determine the type of wastes, their disposition, and cost associated with the waste disposition.

A performance evaluation sample was collected and analyzed at the laboratory (ER-DAR-681). This evaluation is designed to test the performance of the radiological laboratory analysis by submitting a sample with known activity levels.

The conditions for collection of samples for gamma screen analysis were revised (ER-DAR-684). Prior to approval of this DAR samples for the gamma screen analysis were collected and analyzed for shipping purposes. This change, implemented as a cost saving measure, allows for a radiation control technician to use site process knowledge and radiological screening to eliminate the gamma screen prior to shipping.

5.3 CFA-10 Revisions to the Field Sampling Plan

Data were collected during the July 1998 sampling activity to determine the concentration of lead at a depth of 0.6 m (2 ft) and to determine the waste status of the soil.

5.4 Risk Assessment Deviations from the OU 4-13 RI/FS Work Plan

5.4.1 Contaminant Screening

An initial contaminant screen was conducted in Section 3.4 of the OU 4-13 RI/FS Work Plan for each of the retained sites to identify contaminants of potential concern (COPCs). As discussed in the Work Plan, the identified COPCs were to be retained for evaluation in this RI/BRA.

A deviation from this approach is that a supplemental contaminant screen was also conducted for each of the chemicals identified initially as COPCs in the Work Plan. The supplemental contaminant screen (Section 4) was conducted to refine the results of the initial contaminant screen presented in the OU 4-13 RI/FS Work Plan (i.e., refine the list of COPCs to be retained for risk evaluation in the BRA). The supplemental contaminant screen was necessary for the following reasons:

- Removal actions were performed at some of the retained sites (i.e., CFA-06, CFA-13, CFA-15, CFA-17, CFA-42, CFA-47). Additional analytical data was therefore available for these sites following confirmatory soil sampling (i.e., post-removal verification data).

- Additional site characterization of CFA-04 and CFA-08 during the 1997 field season was performed after the initial contaminant screen had been.

- More recent risk-based screening concentrations have been issued since the Work Plan was written. All site and COPCs retained based on the OU 4-13 RI/FS Work Plan were re-screened using the more recent risk-based screening concentrations.

A result of conducting the supplemental contaminant screen is that some of the chemicals that were identified as COPCs in the Work Plan were eliminated, based on the supplemental screen, from risk evaluation in the BRA. These chemicals are summarized in Table 5-1.

In addition, several sites for which COPCs were identified in the Work Plan were eliminated from further evaluation (i.e., CFA-06, CFA-43, CFA-44, CFA-49, CFA-51). These sites were not retained for further evaluation because the supplemental contaminant screen eliminated all COPCs.

5.4.2 Radionuclide Screening Concentrations

The risk-based screening concentrations used to screen radionuclides in the supplemental contaminant screen differ from the risk-based radionuclide screening concentrations used in the RI/FS Work Plan. Risk-based radionuclide screening concentrations used in the supplemental contaminant screen were based on residential 100-year values presented in Table 5 of "Radionuclide Risk-Based Concentration Tables" (Fromm 1996).

Table 5-1. COPCs identified in the Work Plan that were eliminated in the supplemental contaminant screen and not evaluated in the BRA

Site	COPC Not Evaluated
CFA-04	Aroclor-1254, Carbazole, Lead
CFA-06	Arsenic, Lead
CFA-07	Arsenic, Co-60
CFA-08	Aroclor-1254, Arolcor-1260, Arsenic, Carbazole, Isophorone, Am-241, Co-60, Eu-152, Eu-154
CFA-10	Aroclor-1254, Aroclor-1260, Arsenic
CFA-12	Co-60, Cs-134, Eu-154, Zn-65
CFA-17	Aroclor-1260, Arsenic, Benzo(b)fluoranthene, Lead
CFA-42	2-Methylnaphthalene
CFA-43	Lead
CFA-44	Lead
CFA-46	TPH-g
CFA-47	Benzo(b)fluoranthene, Chrysene
CFA-49 (Evaluated as CFA-08 STP)	Co-60
CFA-51	Lead

5.5 Revisions to the WAG 4 Miscellaneous Sites 1997 Non-Time Critical Removal Action. The DARs discussed incorporated changes to the FSP (DOE 1997).

Revisions to the FSP were made during the removal action which are documented with DARs. Changes to the FSP were necessary due to the varying conditions encountered at the removal action sites. The presence of structures such as buildings, tanks, and drywells required a flexible approach to field sampling. The changes to the FSP included the addition of sample locations and analyses intended to ensure that contaminants were located and removed as necessary. The addition of screening samples resulted in less standby or down- time for workers and equipment at the sites.

ER-DAR-770

Six soil samples and associated QA/QC samples were added to the plan for collection at CFA-17 and CFA-47. These samples were collected to meet the requirements in the Risk Based Corrective Action guidance document (Idaho 1997). Samples were analyzed to determine physical properties, including dry bulk density, porosity, moisture content, total organic content, and hydraulic conductivity.

ER-DAR-922 (9/12/97) and –948 (9/25/97)

Sample locations were added to the FSP to collect screening samples from the subsurface soils in the vicinity of former building CFA-640. The number and location of samples was determined by the field team leader based on the presence of visible contamination or the type of structures found. Samples were analyzed for PAHs, VOCs, and metals.

ER-DAR-904 (8/20/97)

Ten screening locations (maximum) were added to the FSP to collect samples from locations at CFA-42. The number and location of samples was determined by the field team leader based on the presence of contamination found during demolition of structures at the site. Samples were analyzed for PAHs and VOCs.

ER-DAR-855, -971, and -976

Additional samples were added to the plan to collect screening samples at CFA-13, -15, -17, -42, and –47. Screening samples were collected to direct excavation of contaminated soils. The number and location of samples were determined by the field team leader based on the presence of potential contamination found at the sites. Samples were analyzed for PAHs, metals, and VOCs.

5.6 References

Fromm, Jeff, 1996. Radionuclide Risk-Based Concentration Tables. Memo to INEL WAG Managers and Technical Support Staff, January 3.

Idaho, 1996, *Risk Based Corrective Action Guidance Document for Petroleum Releases,* 1997, Idaho DEQ-RBCA, August.

McCormick, S. H., P. J. Jessmore, J. M. Schafer et al., 1997, S. M. Rood, J. E. Stephan, and R. L. Vanhorn, 1997, *Work Plan for Waste Area Group 4 Operable Unit 4-13 Comprehensive Remedial Investigation/Feasibility Study*, DOE/ID-10550, March.

Wells, R., 1997, *Field Sampling Plan for the WAG 4 Miscellaneous Sites 1997 Non-Time-Critical Removal Action (CFA-13, -15, -17, -47, and -42)*, DOE/ID-10583, Revision 0, May.

6. HUMAN HEALTH BASELINE RISK ASSESSMENT

The WAG 4 BRA is the first of a two part evaluation. The second part is the ecological risk assessment (ERA) (see Section 7). The human health risk assessment approach used in the BRA is based on the *Risk Assessment Guidance for Superfund (RAGS),* (EPA 1989a), the *INEL Track 2 Guidance Document* (DOE-ID 1994), and the *INEL Cumulative Risk Assessment Guidance Protocol* (LMITCO 1995).

Preliminary evaluations of both human health and ecological risks at WAG 4 have been completed as part of the *Work Plan for Waste Area Group 4 Operable Unit 4-13 Comprehensive Remedial Investigation Study* (DOE-ID 1997). The WAG 4 Human Health Contaminant Screening (Section 3.4 of the OU 4-13 Work Plan) was developed as a preliminary evaluation of WAG 4 human health risks.

A discussion of general comprehensive risk assessment methodologies is presented in the *INEL Cumulative Risk Assessment Guidance Protocol* (LMITCO 1995). The analysis methods used in INEEL comprehensive risk assessments are often different from the analysis methods used in *INEL Track 1 and Track 2 Risk Assessments* (DOE-ID 1994). The differences between the two types of analyses are present because comprehensive risk assessments analyze risks produced by multiple release sites within a WAG, while Track 1 and Track 2 risk assessments analyze risks from one release site at a time.

To satisfy the broader objective of INEEL comprehensive risk assessments, the *INEL Cumulative Risk Assessment Guidance Protocol* recommends analyzing risks produced through the air and groundwater exposure pathways in a "cumulative" manner. A cumulative analysis of these two exposure pathways involves calculating one WAG-wide risk number for each contaminant of potential concern (COPC) for each air and groundwater exposure route (e.g., inhalation of fugitive dust, ingestion of groundwater, etc.). Analyzing the air and groundwater pathways in a cumulative manner is necessary because contamination from all release sites within a WAG may affect air and groundwater exposure pathways at the WAG. Conversely, individual release sites within a WAG are typically isolated from one another with respect to the soil pathway exposure routes (e.g., ingestion of soil, ingestion of homegrown produce, etc.). As a result, the guidance protocol recommends analyzing soil pathway exposures on a release-site-specific, or "noncumulative" basis in INEEL comprehensive risk assessments.

The details of the "comprehensive" and "cumulative" aspects of the WAG 4 BRA are discussed in more detail in the following sections. In general, the BRA is "comprehensive" because it evaluates risks from all known and potential release sites within WAG 4, and it is "cumulative" because risks from multiple release sites are evaluated for the air and groundwater exposure pathways.

The term "risk" is used throughout this section in a generic sense. Generally the term is used to refer to the possibility of adverse health effects from either carcinogenic or noncarcinogenic contaminants, however, it is also used when only carcinogenic health effects are being discussed. The terms "noncancer risk," "hazard quotient" (HQ), and "hazard index" (HI) are used only when noncarcinogenic health effects are discussed.

6.1 Baseline Risk Assessment Tasks

The tasks associated with development of the WAG 4 human health BRA are activities as follows:

- Data evaluation, including site and contaminant screening

- Exposure assessment
- Toxicity assessment
- Risk characterization
- Uncertainty analysis

These tasks are described in the following subsections.

6.1.1 Perform Data Evaluation

All analytical data collected to date at WAG 4 release sites (see Section 4 for a discussion of the various WAG 4 investigations) were evaluated to determine whether the data are appropriate and adequate for use in the BRA. This evaluation was conducted in accordance with *EPA's Guidance for Data Usability in Risk Assessment* (EPA 1992a). As part of this analysis, sampling data sets were assumed to have lognormal distributions in accordance with *EPA's Guidance on Calculating Concentration Terms* (EPA 1992b); however, statistical distributions for the data were not determined.

The data evaluation tasks that were completed as part of the BRA are as follows:

- Screen of release sites to identify sites that have the potential to produce adverse human health and ecological impacts (see Section 6.2.1 for a discussion of the site screening process)
- Review of available sampling data for the retained release sites. This review included a "process knowledge" evaluation designed to identify any contaminants that may have been released at a given site but not sampled for
- Identification of contaminants detected at each retained release site and screened to identify COPCs (see Section 6.2.2 for a discussion of the contaminant screening process)
- Identification of potential exposure routes for each COPC
- Development of data set for use in the risk assessment.

The results of the data evaluation tasks are presented in Section 6.2.

6.1.2 Conduct Exposure Assessment

The process of exposure assessment quantifies all receptor intakes of COPCs for selected pathways. The assessment consists of estimating the magnitude, frequency, duration, and exposure route of COPCs to humans and ecological receptors. The following exposure assessment tasks were performed as part of the BRA process:

- Identification and characterization of exposed populations
- Identification of complete exposure pathways

- Estimation of contaminant concentrations at points of exposure
  - Soil pathway
  - Air pathway
  - Groundwater pathway
- Estimation of human intake rates
- Calculation of intake factors.

The conceptual site models (CSMs) used to develop the BRA exposure assessment are presented in Figures 6-1 through 6-3, and the results of the exposure assessment tasks are presented in Section 6.3.

6.1.3 Conduct Toxicity Assessment

Toxicity assessment is the process of characterizing the relationship between the dose or intake of a substance and the incidence of an adverse effect in the exposed population. Toxicity assessments evaluate results from studies with laboratory animals, or from human epidemiological studies. These evaluations are used to extrapolate from high levels of exposure, where adverse effects are known to occur, to low levels of environmental exposures, where effects can only be predicted based on statistical probabilities. The results of these extrapolations are used to establish quantitative indicators of toxicity.

Health risks from all routes of exposure are characterized by combining the chemical intake information with numerical indicators of toxicity. These health-protective toxicity criteria are obtained through Environmental Protection Agency (EPA)-developed reference doses (RfDs) or slope factors (SFs). The information used as part of the BRA toxicity assessment is presented in Section 6.4.

6.1.4 Risk Characterization

Risk characterization involves combining the results of the toxicity and exposure assessments to provide a numerical estimate of health risk. This estimate is either a comparison of exposure levels with appropriate toxicity criteria, or an estimate of the lifetime cancer risk associated with a particular intake. Risk characterization also considers the nature and weight of evidence supporting the risk estimate, as well as the magnitude of uncertainty surrounding the estimate. The results of the BRA risk characterization process, including risk estimates for each of the retained release sites, are presented in Section 6.5. Uncertainties associated with risk estimates are presented in Section 6.6.

6.2 Site and Contaminant Screening

This section presents the site and contaminant screening methodologies used in the WAG 4 BRA. These screening methodologies are used to help focus the BRA by identifying release sites and contaminants that do not contribute to the comprehensive human health or ecological risk at WAG 4. The screening methodologies are designed to be conservative so that only sites and contaminants that clearly do not pose any threat of producing adverse human health or ecological effects are identified by the methodologies.

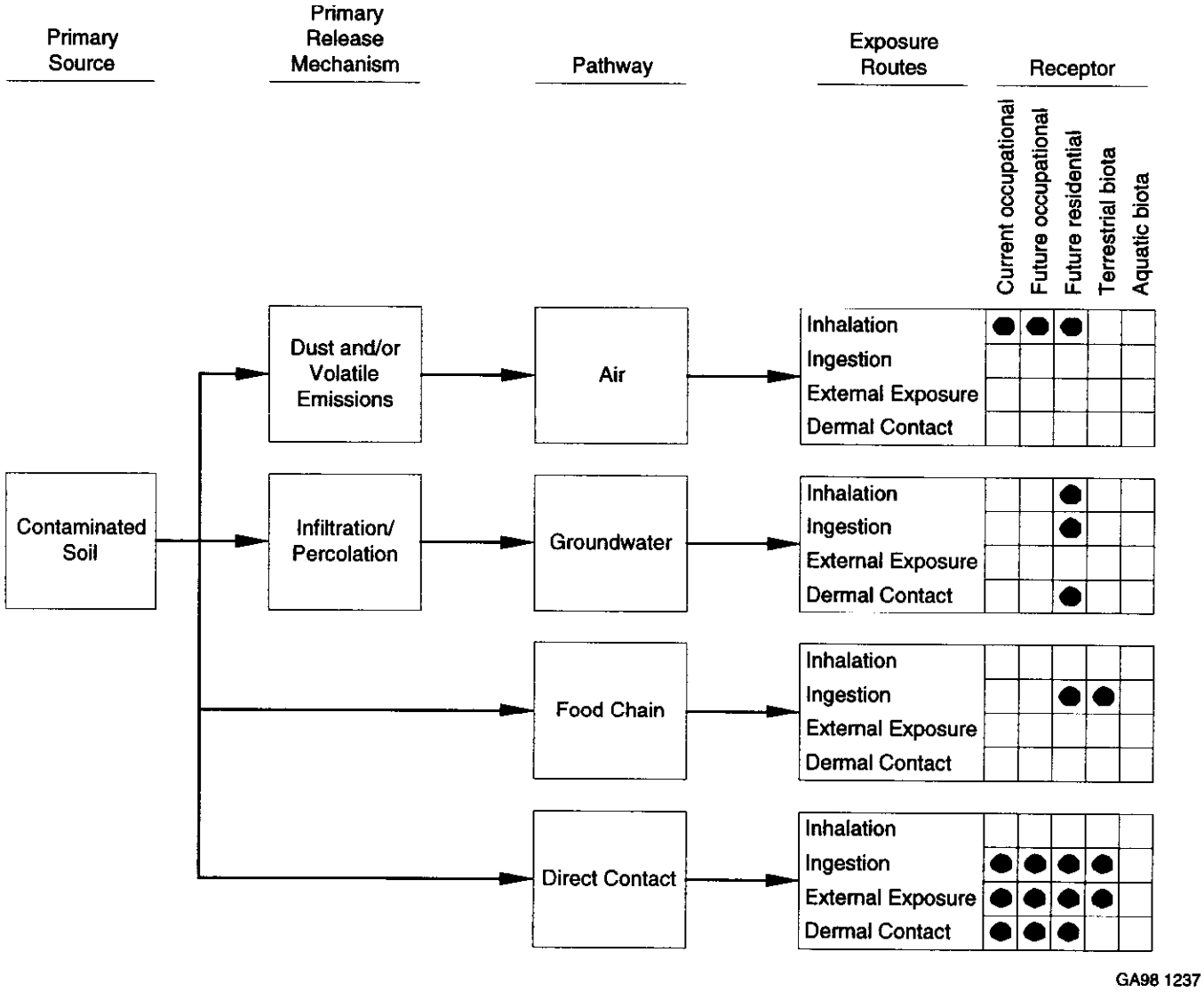


Figure 6-1. Conceptual site model for contaminated surface soil sites at CFA.

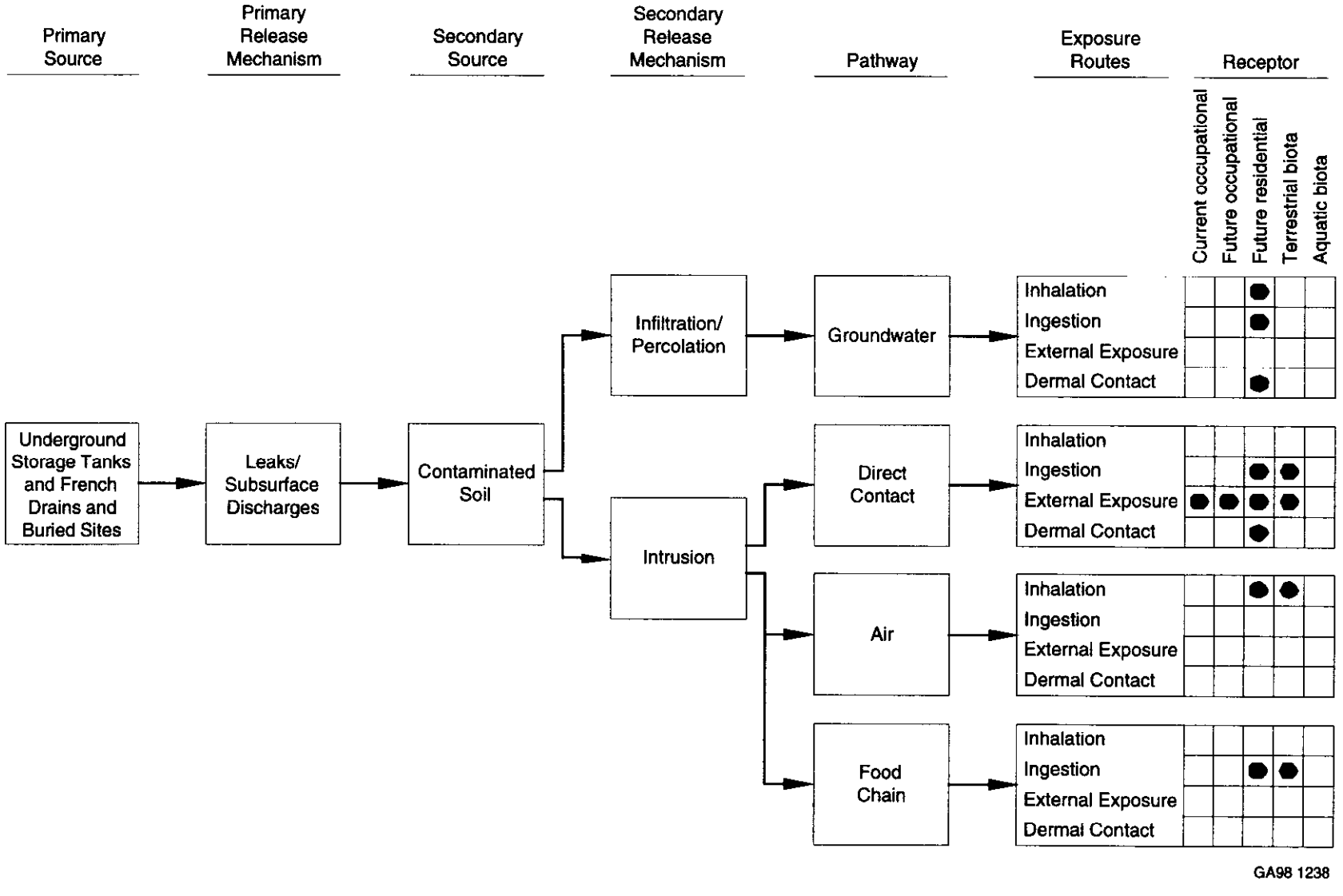


Figure 6-2. Conceptual site model for underground storage tanks and buried waste sites at CFA.

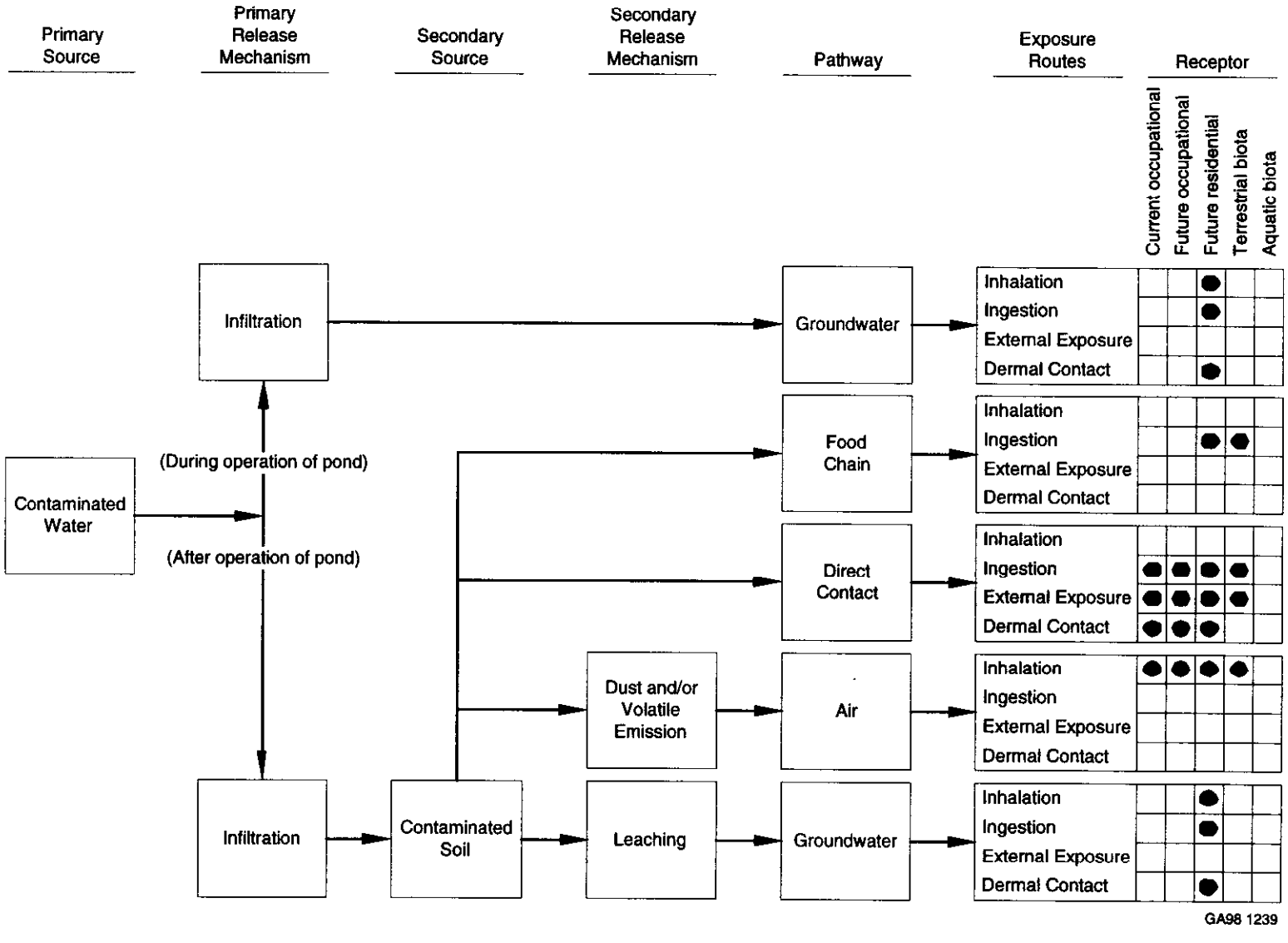


Figure 6-3. Conceptual site model for liquid discharge sites at CFA.

For the remainder of this report, sites and contaminants that pass the screening processes will be referred to as "retained" sites and contaminants; all retained sites and contaminants are further evaluated in Sections 6.3 through 6.6. Likewise, sites and contaminants that fail the screening processes will be referred to as "eliminated" sites and contaminants. All eliminated sites and contaminants require no further evaluation in the BRA.

WAG 4 includes 52 potential hazardous waste release sites such as the landfills, drainage ponds, dry wells, french drains, underground storage tanks, and spill areas. Wastes at these release sites originated from offices, laboratories, maintenance shops, storm and floor drains, and parking lots. Only historic release sites that have been identified at WAG 4 are considered in the OU 4-13 site and contaminant screening processes.

The following sections discuss the site and contaminant screening methodologies. These methodologies are graphically summarized in Figure 6-4.

6.2.1 Site Screening Methodology

Table D-1 presents a list of WAG 4 release sites. All of the sites listed in this table are considered in the site screening process.

The following steps are used to screen release sites:

1. The contaminant sampling information for all WAG 4 sites is compiled.

2. Sites that have not been evaluated by previous risk assessments (i.e., new sites) are identified.

3. Sites that are identified as requiring no further action in the FFA/CO are eliminated.

4. Sites for which a contaminant source does not exist are eliminated. These are sites that have either never contained any contamination, or have had all contamination removed.

5. Sites for which risk was determined to be insignificant by previous risk evaluation activities (e.g., Track 1, Track 2, or other investigations) are eliminated. Risk and HI levels of 1E–06 and 1.0, respectively, are used for this screening step; fewer than 10 release sites are eliminated by this step.

6. Sites containing known contaminants are retained for further evaluation against the contaminant screening criteria.

The site screening steps are discussed in further detail in the following sections.

6.2.1.1 Step 1. The contaminant sampling information for all WAG 4 sites is compiled. In the FFA/CO, WAG 4 is divided into 13 OUs, and these OUs are further divided into individual release sites. Appendix I shows the location of the WAG 4 release sites identified in the FFA/CO. Release site descriptions are presented in Section 4, Nature and Extent of Contamination, and a summary of the site descriptions is presented in Table D-1, Appendix D.

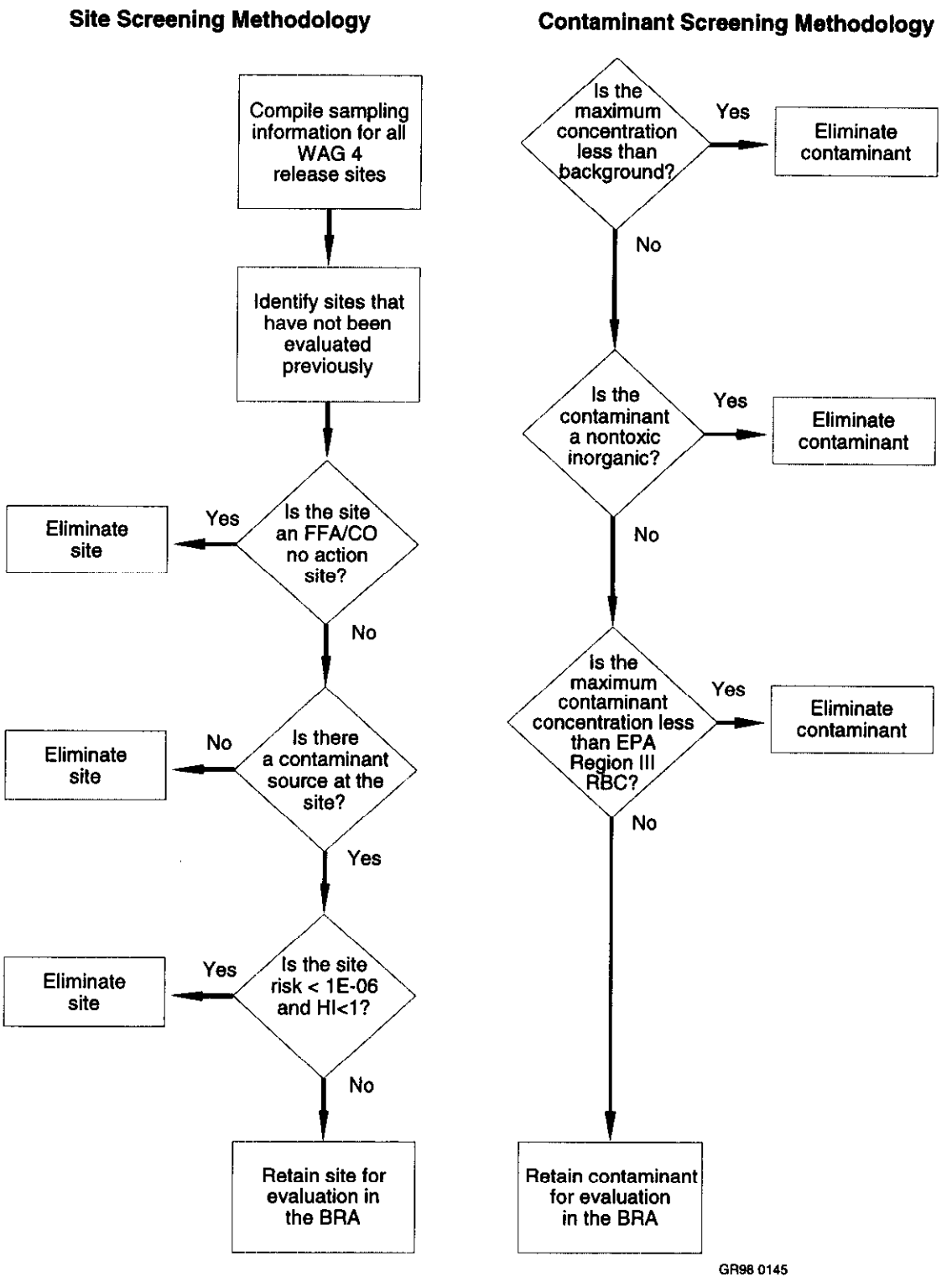


Figure 6-4. Site and contaminant screening methodologies.

6.2.1.2 Step 2. All sites that have not been evaluated by previous risk assessments (for example the Track 1 or Track 2 investigations), (DOE-ID 1994) are identified. In general, only sites that are not listed in the FFA/CO are identified by this screening step. These sites are not subject to Step 5 of the site screening process.

6.2.1.3 Steps 3 through 5. No action, no-source, and low-risk sites are eliminated. Sites that are designated as no action in the FFA/CO, and as a result were not assigned to an OU, are eliminated by Step 3. Sites for which analytical data indicates there is no contaminant sources, or where remedial action has removed all sources, are eliminated in Step 4. Finally, Step 5 eliminates all sites that have been shown to have risks less than 1E-06 and HIs less than 1.0 by Track 1 or Track 2 investigations. Screening criteria of 1E–06 and 1.0 are used because these levels are the minimum acceptable human health risk and HQ values cited in the NCP (see Section 6.5). Of the 52 actual or potential release sites at WAG 4, 34 release sites are screened (i.e., eliminated) in steps 3-5. Table D-2 Appendix D shows which sites have been eliminated from further evaluation, and provides the justification for elimination of those sites.

6.2.1.4 Step 6. All sites that are not eliminated in Steps 3 through 5 of this process are retained for further evaluation (i.e., contaminant screening). These sites are shown in Table D-2, Appendix D.

6.2.2 Contaminant Screening Methodology

Contaminant screening was conducted for all sites that were not eliminated in the site screening process discussed in Section 6.2.1. The contaminant screening methodology is depicted in Figure 6-4. The methodology initially involves compiling all sampling data for each retained site. The sampling results used in the contaminant screening are from various Track 1, Track 2, and other investigation reports, verification sampling following removal actions, characterization data during implementation of the RI/FS Work Plan, and the Integrated Data Environmental Management System (IDEMS) database. The IDEMS database manages INEEL sampling data, and ensures that the data, methods, and data validation qualifiers for all organic, inorganic, and radiological data are consistent.

Two contaminant screens were conducted. Initial contaminant screening was performed as part of the OU 4-13 RI/FS Work Plan, as discussed in Section 6.2.2.1. The purpose of the initial contaminant screening was to make a preliminary determination of COPCs that may require risk evaluation. In addition, as discussed in Section 4, Nature and Extent of Contamination, supplemental contaminant screening was performed in this RI/BRA. The purpose of the supplemental contaminant screen was to refine the results of the initial contaminant screen presented in the OU4-13 RI/FS Work Plan in order to determine which of the retained sites contain COPCs that require quantitative risk evaluation in the RI/BRA. The supplemental contaminant screen was necessary for the following reasons:

- Removal actions were performed at some of the retained sites (i.e., CFA-06, -13, -15, -17/47, -42) after the initial contaminant screen had been conducted. Post-removal analytical data was therefore available for these sites following confirmatory soil sampling.

- Additional site characterization of CFA-04 and -08 was performed after the initial contaminant screening had been conducted. Additional analytical data was therefore available for these sites.

- More recent risk-based screening concentrations (EPA 1997a) have been issued. All sites and COPCs retained based on the OU 4-13 RI/FS Work Plan were re-screened using the more recent risk-based screening concentrations.

The methodologies used to conduct each of these contaminant screens are described below in further detail.

6.2.2.1 RI/FS Work Plan Initial Contaminant Screening. In Section 3.4 of the Work Plan, initial contaminant screening was performed at each of the retained sites to identify COPCs. The following steps were used to screen contaminants in the Work Plan. Each screening step was applied to each contaminant that has been detected at each retained site. As a result of the screening process, individual contaminants may have been eliminated at one retained site, but retained at other sites.

1. Contaminants that are not detected are eliminated from further evaluation.

2. Contaminants that are tentatively identified compounds (TICs) are eliminated from further evaluation. These compounds are discussed in the uncertainty analysis of the BRA (Section 6.6).

3. All contaminants with maximum concentrations that were less than or equal to INEEL background concentrations are eliminated from further evaluation. Background concentrations are taken from *Background Dose Equivalent Rates and Surficial Soil Metal and Radionuclide Concentrations for the Idaho National Engineering Laboratory* (Rood et al. 1995). If a specific background concentration was not available, the contaminant was retained and other screening criteria were considered.

4. Based on EPA guidance (EPA 1991a), six inorganic constituents that are not associated with human toxicity under normal circumstances (aluminum, calcium, iron, magnesium, potassium, and sodium) can routinely be eliminated from analysis in the human health risk assessment. However, these chemicals were retained for analysis in the risk assessment, if the maximum detected concentration was greater than 10 times the background concentration, or if quantitative toxicity information exists.

5. Contaminants that do not exceed the risk-based soil concentrations are eliminated from further evaluation. RBCs are the concentrations that correspond to a calculated lifetime cancer risk of 1E-06, or a HQ of 1. The RBCs used to screen contaminants were calculated using the soil ingestion, soil inhalation, and external exposure pathways. The risk-based screening method was applied by comparing the maximum detected soil concentration for a given contaminant at a given release site against the most restrictive concentration for the contaminant shown in the RBC evaluation.

Chemicals that did not meet the screening criteria outlined above were retained as COPCs for further evaluation in the supplemental contaminant screen, discussed in Section 6.2.2.2. If no COPCs were identified for a retained site using this screen, the site was eliminated from further evaluation.

6.2.2.2 Supplemental Contaminant Screening. Supplemental contaminant screening was performed to refine the results of the initial contaminant screen, based on the availability of new analytical data and the publication of more current risk-based screening concentrations. Only those sites and chemicals that were retained as COPCs as a result of the initial contaminant screen were evaluated in the supplemental contaminant screen. The supplemental contaminant screen was comprised of the following two screening steps:

1. Comparison of the maximum detected contaminant concentration to the respective background concentration.

2. Comparison of the maximum detected contaminant concentration to the respective EPA Region III (1997a) risk-based screening concentration. A contaminant was retained as a COPC if the maximum detected concentration exceeded both screening criteria. Only those contaminants identified as COPCs in the RI/FS Work Plan were included in the supplemental contaminant screen.

The supplemental screens for each of the retained sites are presented in Appendix C. The tables show the maximum concentration of each contaminant found at each retained site, respective background and risk-based screening concentrations, and whether the screening process eliminates a given contaminant. The tables also indicate which of the COPCs are retained for evaluation in the BRA. If no COPCs are identified for a site, the site is eliminated from further evaluation.

The supplemental contaminant screen indicates that COPCs are not present at CFA-26, -46, and -52 to a depth of 3 m (10 ft) bgs. However, these sites are retained for groundwater pathway evaluation in the risk assessment because past activities at these sites resulted in suspected subsurface releases below a depth of 3 m (10 ft) bgs.

The results of the site and contaminant screening process are summarized in Table 6-1. All of the retained sites and COPCs that will be evaluated at those retained sites are listed in this table.

Although removal actions occurred at CFA-13, CF-15, CFA-17/47, CFA-07, CFA-12, and CFA-42, post-removal analytical data indicate that residual contamination still exists at these sites at levels above background or risk-based screening concentrations. However, as discussed in Section 4 (Nature and Extent of Contamination), for each of these sites, residual contamination is only detected in the basalt. Inclusion of these sites for quantitative evaluation in the BRA is conservative because the soil at these sites has already been remediated.

Table D-2 Appendix D indicates which sites were retained based on the supplemental contaminant screen.

6.2.3 Data Uncertainties

There is a possibility that a contaminant may be present at a retained site without being detected in a site investigation. These unidentified contaminants would not be included in the contaminant screening evaluation. The possibility of important contaminants escaping identification is considered small because most site sampling investigations are designed to detect all contaminants that may have been released at a site, and a review of the processes that generated the contamination at each retained site was included as part of the BRA data evaluation process described in Section 6.1.1.

An aspect of the BRA that tends to exaggerate risk results is the evaluation of contaminants with background concentrations that produce calculated risks in excess of 1E–06 (see Section 6.5 for risk characterization methodology). One example of this type of contaminant is arsenic. Arsenic is commonly detected in INEEL soils at concentrations that are slightly higher than the arsenic background screening concentration of 7.4 mg/kg presented in Rood (1995); however, measured concentrations generally are within the range of measured background levels at the INEEL and are therefore likely to be naturally occurring. In addition, arsenic is not associated with known waste producing processes at WAG 4. For these reasons, arsenic was eliminated from further evaluation in the BRA at five sites (i.e., CFA-05, CFA-06, CFA-07, CFA-08, CFA-10). Arsenic is retained as a COPC at CFA-04 because past waste producing activities at CFA-04 may have resulted in concentrating naturally occurring levels of arsenic at the site.

Table 6-1. Summary of WAG 4 Release Sites and COPCs Considered in the BRA.

OU	Site Code	Site Description	COPCs	Contaminated Medium or Media
4-02	CFA-13	Dry Well (South of CFA-640)	Benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)parylene, lead, Am-241, Ra-226, U-235, U-238, Zr-95	Subsurface soil
	CFA-15	Dry Well (CFA-679)	Ra-226	Subsurface soil
4-05	CFA-04	Pond (CFA-674)	Arsenic, mercury, Cs-137, U-234, U-235, U-238	Surface soil and subsurface soil
	CFA-17/47	Fire Department Training Area (bermed) and Fire Station Chemical Disposal	Benzo(g,h,i)perylene, phenanthrene	Subsurface soil
4-07	CFA-07	French Drains E/S (CFA-633)	Ag-108 m, Cs-137, lead, Pu-238	Subsurface soil
	CFA-12	French Drains (2) (CFA-690) [South Drain only]	Ag-108 m, Am-241, Ba-133, Cs-137, Eu-152, U-235, U-238	Subsurface soil
4-08	CFA-08	Sewage Plant (CFA-691), Hot Laundry Drain Pipe (CFA-49) and Drainfield	Cs-137, Pu-239/240, Ra-226, U-235	Surface soil and subsurface soil
4-09	CFA-10	Transformer Yard Oil Spills	Lead	Surface soil
	CFA-26	CFA-760 Pump Station Fuel Spill	Chlorodifluoromethane, phenol, di-n-butylphthalate, TPH-diesel[a]	Subsurface soil
	CFA-42	Tank Farm Pump Station Spills	Phenanthrene	Subsurface soil
	CFA-46	Cafeteria Oil Tank Spill (CFA-721)	Benzene, TPH-diesel, ethylbenzene, toluene, xylenes	Subsurface soil
4-11	CFA-05	Motor Pool Pond	Ac-228, Am-241, Bi-212, Bi-214, Cs-137, lead, Pb-212, Ra-226, Tl-208	Subsurface soil
4-13	CFA-52	Diesel Fuel UST (CFA-730) at Bldg CFA-613 Bunkhouse	Tetrachloroethene; 1,1,1-trichloroethane; TPH-diesel[a]	Subsurface soil

a. Contaminant screening for this site was performed in the OU 4-13 RI/FS Work Plan, a screening table for the site is not included in Appendix C.

6.3 Exposure Assessment

The objective of the human health exposure assessment is to quantify the type and magnitude of potential exposures to human receptors from the COPCs that are present or are migrating from the site. This section outlines the methodologies and assumptions used to calculate the potential daily exposure to each Site COPC. The results of the exposure assessment are combined with chemical-specific toxicity information (Section 6.4) to characterize potential risks posed by WAG 4 COPCs (Section 6.5).

Quantifying receptor intake consists of the following four major steps:

- Identification and characterization of exposed populations
- Evaluation of exposure pathways
- Estimation of contaminant concentrations at points of exposure for the following exposure media:
  - Soil
  - Air
  - Groundwater
- Estimation of contaminant intakes.

Each of these steps is discussed in the following sections.

6.3.1 Identification and Characterization of Exposed Populations

As discussed in the OU 4-13 RI/FS Work Plan, two human receptor populations could potentially be exposed to contaminants found at, or originating from, WAG 4: workers and residents. Potential risks to workers and residents are assessed quantitatively in this BRA. Assumptions associated with evaluating potential exposures and risks to these two receptor populations are discussed in the sections below.
Workers. Because WAG 4 is currently operational, workers at the site are potential receptors. Potential risks to the following two occupational exposure scenarios are assessed in the BRA:

1. A current occupational scenario that lasts for 25 years from the present.
2. A future occupational scenario that starts in 100 years and lasts for 25 years.

Both the current and future occupational scenarios are evaluated assuming radioactive decay. For nonradionuclides, it is conservatively assumed that chemical degradation does not occur; hence the potential risks presented for the future occupational scenario from exposure to nonradionuclides are equivalent to those calculated for the current occupational scenario.

6.3.1.1 Residents. For the purposes of the BRA, residential development is considered as a potential future use of the site, and a future residential exposure scenario is quantitatively evaluated in the BRA.

The residential exposure scenario evaluated in the BRA considers a future resident who moves to the site in 100 years and lives there for 30 years, the reasonable upper-bound residence time (EPA 1991b). Because the nearest single-family residence is currently located several miles from the boundary of WAG 4 and there are no plans for residences to be built at WAG 4, current residents are not evaluated in the BRA.

Groundwater pathway risks are calculated at 100 years in the future for use in the 100-year residential exposure scenario. Groundwater risks for each COPC are also calculated at the time of the maximum groundwater concentration of each COPC, as long as the maximum concentration occurs before 10,000 years in the future. Section 6.3.3.3 presents a more detailed discussion of the groundwater pathway analysis.

The future residential scenario is evaluated assuming radioactive decay for radionuclides. For nonradionuclides, it is conservatively assumed that chemical degradation does not occur.

6.3.1.3 Future Land Use. Future land use at the INEEL will most likely remain industrial. Other potential, but less likely INEEL land uses include agriculture and the return of areas onsite to an undeveloped state. A land use document was developed in an effort to assist DOE in identifying the issues regarding probable future land use (DOE 1996). According to this document, CFA facilities are planned to continue with new development through the 100-year time-frame and will be maintained as a central location for all support functions at the INEEL.

6.3.2 Evaluation of Exposure Pathways

An exposure pathway describes a specific environmental pathway by which a receptor can be exposed to COPCs that are present at or migrating from the site. Five elements comprise an exposure pathway. These elements, shown below, are identified to determine potential exposure pathways at the site:

1. A chemical source
2. A mechanism of chemical release to the environment
3. An environmental transport medium (e.g., air, groundwater) for the released chemical
4. A point of contact between the contaminated medium and the receptor (i.e., the exposure point)
5. An exposure pathway (e.g., ingestion of contaminated groundwater) at the exposure point.

All five of these elements must be met for an exposure pathway to be potentially complete. Information concerning chemical waste sources, chemical release and transport mechanisms, locations of potentially exposed receptors, and potential exposure routes was used to develop a conceptual understanding of the site. This information was summarized schematically in Figures 6-1 through 6-3. In the CSM, potentially complete exposure pathways are designated with a closed circle. Only those exposure pathways deemed to be complete (i.e., where a plausible route of exposure can be demonstrated from the site to the receptor) are quantitatively evaluated in the BRA.

As indicated in the CSM, three categories of sites were retained for evaluation in the BRA as shown in Table 6-2.

Tables 6-3 and 6-4, below, provide a summary of the exposure media and potentially complete exposure pathways associated with these three site types.

6.3.2.1 Occupational Exposure Pathway Assumptions. To evaluate potential occupational risks from exposure to soil, it is assumed that both current and future workers at the sites will only be exposed to contamination from the top 15 cm (6 in) of soil for the soil ingestion, inhalation of fugitive dust and VOC exposure routes. For the evaluation of external radiation exposure, radionuclide activities present in the top 1.2 m (4 ft) of soil will be used. This analysis method is referred to as the occupational nonintrusion exposure scenario, and all occupational exposure scenario analyses in the OU4-13 BRA will include an evaluation of this exposure scenario.

6.3.2.2 Residential Exposure Pathway Assumptions. For the purposes of the BRA, it is assumed that future residents will construct 3 m (10 ft) basements beneath their homes. As a result, all contamination detected in the upper 3 m (10 ft) of each release site will be evaluated for surface pathway exposures. This analysis method will hereafter be referred to as a "residential intrusion scenario," and all residential exposure scenario analysis in the OU 4-13 BRA will include the residential intrusion assumption.

In general, the residential exposure scenario only evaluates adult exposures. The reason for this limitation is that the risk results presented in the BRA are calculated using very conservative exposure assumptions. These assumptions most likely cause the calculated risk results to overestimate the actual risks to even sensitive subpopulations, such as children, that would result from exposure to the site's contamination.

The exception to this rule is associated with the soil ingestion exposure route described in Section 6.3.3.1. Under this exposure route, six years of childhood soil ingestion and 24 years of adult soil ingestion are included in the contamination intake calculation. Soil ingestion is the most critical exposure route for children who may someday live at WAG 4 because of the relatively large amount of soil that children may ingest.

6.3.3 Estimation of COPC Concentrations at Points of Exposure

Exposure point concentrations are one of several parameters required to estimate the intake of chemicals by a human receptor. Exposure point concentrations were calculated in accordance with EPA guidance for calculating concentrations terms (EPA 1992b). The calculated exposure point concentrations correspond to the upper 95 percent confidence limit (95% UCL) of the mean for each of the COPC data sets evaluated. As part of the analysis, all data sets are assumed to have lognormal distribution.

EPA (1989a) risk assessment guidance recommends consideration of the positively detected results together with the non-detected results (i.e., sample quantitation limits). Following this guidance, for all results reported as "non-detect," one-half of the sample quantitation limit was assumed as a conservative proxy concentration for each sample with a non-detect result.

Table 6-2. Retained Site Categories

Site Category	Corresponding Retained Site
Surface Soil	CFA-10 Transformer Yard Oil Spills
	CFA-17 Fire Department Training Area (bermed) and CFA-47 Fire Station Chemical Disposal
UST and Buried Waste	CFA-07 French Drain E/S [CFA-633]
	CFA-12 French Drains [CFA-690], south drain only
	CFA-46 Cafeteria Oil Tank Spill [CFA-721]
	CFA-52 Diesel Fuel UST [CFA-730] at Building CFA-613 Bunkhouse
Liquid Discharge	CFA-04 Pond [CFA-674]
	CFA–05 Motor Pool Pond
	CFA–08 Sewage Plant [CFA-691], Septic Tank [CFA-716], and Drainfield
	CFA-13 Dry Well (South of CFA-640)
	CFA-15 Dry Well (CFA-674)
	CFA–26 CFA-760 Pump Station Fuel Spill
	CFA–42 Tank Farm Pump Station Spills

Table 6-3. Summary of Current and Future Occupational Exposure Media and Pathways

	Occupational Exposure Medium– Occupational Exposure Pathway			
Site Type	Soil – Ingestion	Soil—Dermal Contact	Soil— External Exposure	Air—Inhalation of VOCs and Particulates
Surface Soil	x	x	x	x
UST, Buried Sites			x	
Liquid Discharge	x	x	x	x

x = Potentially complete exposure pathway; will be quantitatively evaluated in the BRA.

Table 6-4. Summary of Future Residential Exposure Media and Pathways

	Residential Exposure Medium—Residential Exposure Pathway							
Site Type	Soil— Ingestion	Soil— Dermal Contact	Soil— External Exposure	Soil – Home Grown Produce Ingestion	Air– Inhalation of VOCs and Particulates	Ground Water– Ingestion	Ground Water— Dermal Contact	Ground Water— Inhalation of VOCs
Surface Soil	x	x	x	x	x	x	x	x
UST, Buried Sites[a]	x	x	x	x	x	x	x	x
Liquid Discharge[a,b]	x	x	x	x	x	x	x	x

x = Potentially complete exposure pathway; will be quantitatively evaluated in the BRA.

[a] Evaluation of CFA-07, CFA-08 STD, CFA-26, CFA-42, CFA-46, and CFA-52 is limited to groundwater pathways because soil contamination is limited to depths greater than 10 ft below ground surface (bgs) (see Sections 4.1.13, 4.1.15, and 4.1.18, respectively).

[b] Evaluation of CFA-05 is limited to groundwater pathways.

If the calculated 95% UCL of a chemical in a medium-specific data set exceeds the maximum concentration detected in that data set, EPA (1989a) recommends that the maximum detected concentration be selected as the exposure point concentration. Exceedance of the maximum detected concentration typically occurs when dilution effects have resulted in reporting of very high sample quantitation limits (i.e., non-detect values) or if a limited number of sample results are available (e.g., less than ten).

Site surface areas and soil volumes, and chemical-specific properties [i.e., molecular weights, radionuclide half-lives, soil to water partition coefficients (k_d), solubilities, octanol-water partition coefficients (K_{ow}), organic carbon partition coefficients (K_{oc}), diffusivities, Henry's Law Constants, and plant uptake factors (PUFs)] are required to estimate exposure point concentrations. Table D-3 summarizes the surface areas and soil volumes that were used to calculate COPC exposure point concentrations. Table D-4 provides a summary of chemical-specific property values that were used to calculate COPC exposure point concentrations.

The depths of contamination used to evaluate the identified potentially complete soil exposure pathways are based on guidance given in the *INEL Track-2 Investigation Manual* (DOE-ID 1994). Contaminant exposure point concentrations for soil are calculated for a range of depth intervals to evaluate the different exposure scenarios and pathways, as shown below.

Depth Interval	Exposure Scenario and Pathway(s)
0 to 0.15 m (0 to 6 in.)	Occupational scenario: soil ingestion, inhalation of fugitive dust, inhalation of volatiles
0 to 1.2 m (0 to 4 ft)	Occupational scenario: external radiation exposure
0 to 3 m (0 to 10 ft)	Residential scenario: all soil pathway and air pathway exposure routes
All sample results included, regardless of depth	Residential scenario: all groundwater pathway exposure routes

For each of these soil depth intervals, 95% UCL concentrations were calculated for each COPC based on the methodology described above. The calculated soil concentrations are summarized in Tables D-5 through D-7 by depth interval for each COPC.

The concentration values shown in Tables D-5 through D-7 indicate that a given COPC was detected within the depth interval shown in the table, not that the COPC contamination extends to the bottom of the interval. For example, mercury could have a calculated 0-to-3-m (0-to-10-ft) concentration at a given site even if the site's mercury contamination only extends from 0 to 1.5 m (0 to 5 ft).

The exposure point concentrations for each of the above depth intervals were calculated by volume-weighting 95% UCL concentrations for each of the depth intervals. For example, the 0-to-1.2-m (0-to-4-ft) exposure point concentrations were calculated by determining 95% UCL contaminant concentrations that correspond to soil depths of 0 to 0.5 ft bgs and 0.5 to 4.0 ft bgs. The 95% UCL concentrations for those two soil depth ranges were then volume-weighted using associated depths (i.e., 0.5 ft, 3.5 ft) to calculate a volume-weighted exposure point concentration for the 0–4 ft bgs depth interval. The example algorithm below shows how the exposure point concentration for the 0–4 ft bgs depth interval is calculated.

$$\text{Exposure Point Concentration (0-4 ft)} = \frac{(95\%UCL_{0-0.5\,ft} \times 0.5) + (95\%UCL_{0.5-4.0\,ft} \times 3.5)}{4}$$

Assumptions for the depth of COPC vertical migration are presented in Section 4, Nature and Extent of Contamination. For COPCs that are detected at depths greater than 10 ft bgs, the maximum depth used to calculate exposure point concentrations is based on the assumed vertical migration depth. For example, if mercury is detected at 12 ft bgs but is assumed to migrate 10 ft to a depth of 22 ft bgs, then the 95% UCL calculated for mercury for the > 10 ft depth interval is used to represent mercury concentrations from 10 to 22 ft bgs. Calculation of the exposure point concentration for groundwater pathways would then be based on the following algorithm:

$$\text{EPC (0 - >10 ft)} = \frac{(95\%UCL_{0-0.5\,ft} \times 0.5) + (95\%UCL_{0.5-4\,ft} \times 3.5) + (95\%UCL_{4-10\,ft} \times 6) + (95\%UCL_{10-22\,ft} \times 12)}{22}$$

Volume weighted averaging has the potential for producing underestimation of exposure point concentrations at sites that only shallow subsurface contamination (e.g., CFA-10). This potential underestimation would occur if a future receptor were only exposed to the shallow surface soils at the contaminated site, instead of being exposed to soil down to a depth of 3 m (10 ft) bgs. The potential underestimation can be corrected for by multiplying the risk results for a given contaminant at an affected site by the ratio of the contaminant's 0–0.5 ft concentration to its 0–10 ft concentration.

As discussed in Section 6.3.1, for each of the exposure scenarios evaluated, radioactive decay is assumed to occur over the exposure period evaluated. Radioactive decay is estimated using the following equation:

$$C = Co\, e^{-8t} \qquad (6\text{-}1)$$

where

C = concentration at time = t (pCi/g)

Co = initial concentration at time = 0 (pCi/g)

8 = decay constant

t = time interval (years)

The decay constant (8) is calculated using the contaminant half-life ($t_{1/2}$) in the following equation:

$$8 = \ln 2 / t_{1/2} \qquad (6\text{-}2)$$

where

ln2 = natural log of 2

$t_{1/2}$ = half-life of the radionuclide (years)

By substituting 8 in the first equation, equation (6-1) becomes:

$$C = Co\, e^{-(\ln 2 / t1/2)\, t} \qquad (6\text{-}3)$$

This equation accounts for radioactive decay by estimating the radionuclide concentration at the start of a given exposure, and then calculating the average concentration during the length of the scenario. For example, the concentration of a given radionuclide analyzed in the current occupational exposure scenario is the average concentration that would exist between 0 and 25 years in the future, and the concentration analyzed in the 100-year future residential scenario is the average concentration that would exist between 100 and 130 years in the future. To calculate that average concentration for the future residential scenario, equation (6-3) must be integrated between the start time (t = 100 years) and end time (t = 130). The integral of equation (6-3) is as follows for the residential scenario:

$$C_{average} = C_o \times \frac{\left\{e^{-[(\ln 2 / t1/2)\times 100]} - e^{-[(\ln 2 / t1/2)\times 130]}\right\}}{\left[\frac{\ln 2}{t1/2} \times (130 - 100)\right]}$$

The average radionuclide concentrations over each exposure period evaluated are shown in Tables D-8 through D-12b. These concentrations are used in the intake calculations for each exposure pathway.

The effects of radioactive progeny are only considered by using "+D" SFs in the radionuclide risk calculations (see Section 6.5). Decay and in growth calculations are not performed for complete radionuclide decay chains. The use of "+D" SFs account for risks produced by daughter products that are in secular equilibrium with their parent radionuclides (EPA 1995a).

The following sections describe the methodology used to calculate soil, air, and groundwater exposure point concentrations for the identified COPCs.

6.3.3.1 Soil Exposure Pathway Methodology. The following soil exposure routes are identified in the CSM (Figures 6-1 through 6-3) as potentially complete for the residential and/or occupational exposure scenarios:

- Soil ingestion
- External radiation exposure
- Dermal absorption from soil
- Ingestion of homegrown produce (residential scenario only)

The following sections describe the methodology used to calculate soil exposure point concentrations for these exposure routes. The calculated exposure point concentrations are used to estimate potential exposures from these exposure routes.

6.3.3.1.1 Soil Ingestion—Because exposures through the soil pathway are not likely to occur from more than one release site at a time, the soil pathway is evaluated on a site-by-site basis.

As with the air pathway, soil pathway risks and HQs are calculated at 0 and 100 years in the future for the occupational exposure scenario, and at 100 years for the residential scenario.

6.3.3.1.2 Homegrown Produce Ingestion—The homegrown produce ingestion exposure route includes an evaluation of COPC concentrations in plants caused by both root uptake and irrigation with contaminated groundwater. At each retained site, the total source concentration evaluated in the homegrown produce ingestion exposure route is calculated by combining the 95% UCL on the mean concentration for a given COPC (or the maximum concentration if the maximum is less than the 95% UCL) with the soil concentration that would result from equilibrium partitioning between soil and groundwater contaminated with the COPC.

Homegrown produce concentrations assumed for each COPC are presented in Table D-13. To evaluate the average soil concentration of radioactive COPCs in soil when irrigating with groundwater, the integrated form of Equation 5.39 in *Nuclear Regulatory Commission (NRC) Guidance Document* (NRC 1993) is used:

$$C_S(t) = \frac{\frac{\dot{I}_v}{L_i+\lambda}\left(t_e + \frac{e^{-(L_i+\lambda)t_e}}{L_i+\lambda}\right) + \frac{C_{so}}{L_i+\lambda}\left(1 - e^{-(L_i+\lambda)t_e}\right) - \frac{\dot{I}_v}{(L_i+\lambda)^2}}{t_e} \quad (6\text{-}5)$$

where

$C_s(t)$ = the average concentration of a COPC in soil for the exposure period, t_e (pCi/g)

$\dot{I}_v$ = COPC input rate from irrigation (pCi/g-day)

L_i = leach rate constant $(day)^{-1}$

t_e = exposure period [10,950 days (30 years × 365 days/year)]

C_{so} = average concentration of COPC in the top 3 m (10 ft) of soil at the start of the residential exposure period (pCi/g)

8 = $\ln 2 / t_{1/2}$ where $t_{1/2}$ is the half-life of the radionuclide expressed in days

For nonradioactive COPCs, this equation reduces to the following:

$$C_s(t) = \frac{\frac{\dot{I}_v}{L_i}\left(t_e + \frac{e^{-(L_i t_e)}}{L_i}\right) + \frac{C_{so}}{L_i}\left(1 - e^{-(L_i t_e)}\right) - \frac{\dot{I}_v}{L_i^2}}{t_e} \quad (6\text{-}6)$$

The COPC input rate from irrigation is given by the following equation:

$$\dot{I}_V = C_W \times \frac{I_R}{\rho \times T} \quad (6\text{-}7)$$

where

$\acute{I}_v$ = COPC input rate from irrigation (mg/g-day or pCi/g-day)

C_W = average concentration of a COPC in groundwater for the exposure period (mg/L or pCi/L)

I_R = irrigation rate (8.47 L/m^2-day × 90 days/365 yrs) (Maheras et al. 1994)

ρ = soil density (1.5E+06 g/m^3)

T = thickness of root zone (0.2 m) (7 in.) [International Atomic Energy Agency (IAEA) 1994].

The leach rate constant is given by the following equation (Baes and Sharp 1983):

$$L_i = \frac{P}{\theta_c \times \left(1 + \frac{K_d \times \rho}{\theta_c}\right) \times T} \times CF \qquad (6\text{-}8)$$

where

L_i = leach rate constant (day)$^{-1}$

P = net water percolation rate (0.86 m/1 year) [infiltration rate of 0.1 m/1 year, as presented in *INEL Track 2 Guidance* (DOE-ID 1994), plus the contribution from irrigation]

θ_c = volumetric water content in source volume (0.41 m^3/m^3) (Rood 1994)

K_d = COPC-specific soil-to-water partition coefficient (cm^3/g)

ρ = soil density (1.5 g/cm^3)

T = thickness of root zone (0.2 m) (IAEA 1994)

CF = conversion factor (1 year/365 days).

Finally, concentrations of COPCs in affected homegrown produce are calculated using the following equation (EPA 1995b):

$$C_p(t) = C_s(t) \times B_v \qquad (6\text{-}9)$$

where

Cp(t) = average concentration of a COPC in homegrown produce from root uptake (pCi/g or mg/kg)

Cs(t) = average concentration of a COPC in soil for the exposure period (pCi/g or mg/kg)

Bv = COPC-specific soil-to-plant uptake coefficient (mass of COPC/dry mass of plant material per mass of COPC/dry mass of soil).

Homegrown produce contaminant concentrations calculated using the above equations are presented in Table D-13.

6.3.3.1.3 External Radiation Exposure—For the external radiation exposure route, standard EPA protocols are used to estimate risks for all retained sites. In other words, external radiation exposure risks are calculated by multiplying radiation intakes for specific isotopes by the radionuclide slope factors presented in EPA's Health Effects Assessment Summary Tables (HEAST) (EPA 1995a). The standard EPA protocols are used because all of the retained sites in the BRA have radionuclide contamination that is at least 0.2 m (6 in.) thick over a large area. This thickness is large enough to satisfy the assumption that an increase in source thickness will not cause an increase in surface radiation exposures.

6.3.3.1.4 Dermal Exposure—Similarly to the soil ingestion exposure route, dermal exposure to soil is not likely to occur from more than one release site at a time. Therefore, dermal exposure to soil is evaluated on a site-by-site basis.

Potential risks from dermal absorption from soil are based on the potential for a chemical to be absorbed through skin. This potential is quantified by chemical-specific absorption factors (ABS) (i.e., the fraction of a chemical that may be absorbed through skin).

ABS values are not well quantified for many of the chemicals that have been detected at WAG 4. In the absence of this chemical-specific information, EPA Region III has issued general guidelines for evaluating dermal exposure. These guidelines include recommendations on default ABS values in the absence of chemical-specific values (EPA 1995c). Based on EPA (1995c), organic chemicals generally have relatively high ABS values and therefore have the greatest potential for being absorbed through the skin. To evaluate potential dermal exposures from contact with volatile organic chemicals (VOCs), EPA (1995c) recommends assuming an ABS value of three percent, or 0.03, for VOCs with vapor pressures (VP) lower than the VP of benzene (95.2 mm Hg). For VOCs with a VP greater than 95.2 mm Hg, an ABS of 0.05 percent, or 0.0005, should be assumed. For semi-volatile organic chemicals (VOCs), EPA (1995c) recommends use of an ABS of 10 percent, or 0.10.

Dermal uptake is generally not an important route of uptake for metals or radionuclides, which have small dermal absorption and dermal permeability constants; therefore, this BRA does not include an evaluation of potential risks from dermal absorption of metals and radionuclides in soil and groundwater. An exception to this rule is the evaluation of potential risks from dermal exposure to arsenic in soil and groundwater. Arsenic is retained as a soil COPC at CFA-04. The ABS recommended for arsenic is three percent, or 0.03 (EPA 1995). This ABS value is relatively high; therefore, arsenic is included with organics in the evaluation of potential risks from dermal contact with soil at CFA-04.

Modeling of arsenic to future residential receptor well locations (see Section 6.3.3.3, Groundwater Exposure Pathway) is also conducted to because arsenic is retained as a soil COPC at CFA-04. If results of the groundwater modeling indicate that arsenic is expected to reach future residential receptor well locations, then arsenic will also be included in the groundwater cumulative risk analysis, and potential risks posed by dermal contact with arsenic in groundwater will be evaluated.

6.3.3.1.5 Soil Exposure Pathway Assumptions—The evaluation of potential exposures from soil pathways is based on the following assumptions:

- Soil pathway exposures from multiple release sites are insignificant (see Section 6, Uncertainty Analysis).
- The likelihood that a future resident will raise meat and dairy products on a residential lot at WAG 4 is assumed to be negligible, in accordance with INEEL guidance on analysis of the homegrown produce ingestion exposure route (LMITCO 1996). As a result, risks from the ingestion of meat and dairy products are not quantitatively evaluated in the BRA.
- A receptor is assumed to be present at each retained site for the full exposure duration (30 years for a residential receptor and 25 years for an occupational receptor).

6.3.3.2 Air Exposure Pathway Methodology. The following air exposure pathways are identified in the CSM (Figures 6-1 through 6-3) as potentially complete for the residential and/or occupational exposure scenarios:

- Inhalation of fugitive dust
- Inhalation of volatiles.

Because there is a possibility that contamination from multiple sites can mix together within the air volume above WAG 4, the air pathway is analyzed in a cumulative manner in the WAG 4 BRA. To perform this cumulative analysis, a WAG-wide average soil concentration is calculated for each COPC. The concentration of each COPC in the respirable particulate matter above WAG 4 is assumed to equal this average soil concentration. Averaging contaminant concentrations above WAG 4 for the air pathway produces one contaminant-specific risk estimate for each air pathway exposure route [i.e., for each time period, each air pathway exposure route has the same risk or HI at every retained site (see Section 6.5)].

The equations discussed below will be used to estimate airborne contaminant concentrations:

$$C_{air} = CF \times R \times C_{soil} \qquad (6\text{-}10)$$

where

C_{air} = contaminant concentration in air (mg/m^3 or pCi/m^3)

CF = conversion from kg to mg for nonradionuclides or g to mg for radionuclides

R = airborne respirable particulate matter concentration (0.011 mg/m^3). This value is given in Appendix B of the *INEL Site Environmental Monitoring Reports* (e.g., Hoff et al. 1993), and represents the arithmetic mean, of weekly airborne respirable particulate matter concentrations by the TAN low volume air sampling station

C_{soil} = WAG average contaminant soil concentration (mg/kg or pCi/g) weighted by site area.

and

$$C_{soil} = \frac{\Sigma\ C_n\ A_n}{A_T} \qquad (6\text{-}11)$$

where

C_n = contaminant soil concentration at site *n* (mg/kg or pCi/g)

A_n = surface area of site *n* (m^2)

A_T = total area of the WAG 4 retained sites (m^2) for which non-volatile contaminants are present in the top 0.15 m (0.5 ft) and 3.05 m (10 ft), respectively, for the occupational and residential exposure scenarios

n = number of retained sites.

The equation used for estimating concentrations of airborne volatiles is as follows:

$$C_{air} = \frac{\Sigma\ (C_n\ /\ VF_n)\ A_n}{A_T} \qquad (6\text{-}12)$$

where

C_n = contaminant soil concentration at site n (mg/kg)

VF_n = volatilization factor [as described in *INEL Track 2 Guidance* (DOE-ID 1994)] for site *n* (m^3/kg)

A_n = surface area of site *n* (m^2)

A_T = total area of the WAG 4 retained sites (m^2) for which volatile contaminants are present in the top 0.15 m (0.5 ft) and 3.05 m (10 ft), respectively, for the occupational and residential exposure scenarios

These equations produce conservatively high estimates of airborne COPC concentrations because no credit is taken for dilution of airborne concentrations caused by dust blown from uncontaminated areas of the WAG.

As with the soil pathway analysis, the air pathway receptor is either a current occupational worker (who is assumed to be exposed for 25 years) or a hypothetical future resident (who is exposed for 30 years). Air pathway risks and HQs are calculated at 0 and 100 years in the future for the occupational scenario, and at 100 years in the future for the residential scenario. Estimated concentrations of COPCs in fugitive dust and estimated concentrations of volatiles for each exposure period are presented in Tables D-15 through D-17.

6.3.3.2.1 Air Exposure Pathway Assumptions—The evaluation of potential exposures from air pathways is based on the following assumptions:

- The concentration of each retained contaminant in the respirable particulate matter above the WAG will be equal to each contaminant's WAG wide average soil concentration.

- The airborne concentration of each retained contaminant will be the same at every point inside the WAG boundaries.

- The air pathway receptor will be assumed to spend the entire exposure duration (25 years for current occupational workers and 30 years for future residents) working or living within the boundaries of the WAG.

6.3.3.3 Groundwater Exposure Pathway. To quantify potential risks from exposure via groundwater pathways, modeling of contaminant concentrations in groundwater is required. For the groundwater pathway analysis, every contaminant that is not eliminated by the contaminant screening process (described in Section 6.2) is assumed to have the potential for migrating to groundwater, but only manmade sources of contamination are considered in the analysis. The following groundwater exposure pathways are identified in the CSM (Figures 6-1 through 6-3) as potentially complete for the future residential exposure scenario:

- Ingestion of groundwater

- Dermal absorption of groundwater

- Inhalation of volatiles produced by indoor use of groundwater.

WAG 4 includes surface or buried sources of potential groundwater contamination. Precipitation, infiltrating the subsurface and passing through these surface and near surface contaminated soils, can leach contamination to the aquifer beneath WAG 4.

Groundwater concentrations resulting from surface and near surface sources are estimated using the computer code GWSCREEN (Rood 1994). For each COPC, GWSCREEN produces groundwater concentrations versus time as the code output. From this output, the maximum 30-year average groundwater concentration of each COPC, and the 30-year average concentrations at 100 years in the future, are calculated. The average concentrations at year 100 are used to calculate groundwater pathway risks for the residential exposure scenario, and the maximum average concentrations are used to calculate maximum expected groundwater risks.

The total mass of each contaminant considered in the GWSCREEN modeling is calculated by summing the contaminant masses from the retained sites. The contaminant mass at each retained site is derived by multiplying the representative average soil concentration for each contaminant (or maximum detected soil concentration if paucity of data precludes preparation of a volume- and depth-weighted average concentration) by the mass of contaminated soil at the site. For example, if a contaminant has a volume- and depth-weighted average concentration of 7 mg/kg at a site with dimensions of $10 \times 10 \times 1$ m ($30 \times 30 \times 3$ ft), the mass of the contaminant that would be used in the GWSCREEN modeling would be 1.05E+6 mg (i.e., [7mg/kg] × [10m × 10m × 1m] × [1.5 g/cm3] × [1E+06 cm3/m3] × [1E-03 kg/g] = 1.05E+6 mg).

Data used in this groundwater analysis are contained in Table 6-5 through Table 6-11. GWSCREEN input parameters are shown in Table 6-6 through Table 6-8. The COPC masses used in the GWSCREEN modeling are shown in Table 6-9 while resulting groundwater concentrations are presented in Table 6-10 and 6-11.

Table 6-5. Interbed thickness for wells in the vicinity of CFA.

Well	Cumulative Vadose Zone Interbed Thickness (m)	Well	Cumulative Vadose Zone Interbed Thickness (m)
CFA-2	12.8	LF2-09	33.5
LF3-09	16.5	LF3-11	34.6
LF2-12	17.7	LF2-08	36.4
LF2-10	23.2	LF2-11	39.5
LF3-11A	23.2	LF3-10	41.5
LF3-08	30.8	CFA-1	52.1

Table 6-6. GWSCREEN parameters and the values used for transport modeling.

Variable	Parameter Description	Value[a]	Units
L	Source length parallel to aquifer flow direction	site-specific	M
W	Source width perpendicular to aquifer flow direction	site-specific	M
D_s	Thickness of source	site-specific	M
I	Infiltration rate (Darcy flux)	0.1	m/yr
v_a	Aquifer pore velocity	570	m/yr
θ_s	Volumetric water content in source	0.3	Unitless
θ_u	Volumetric water content in unsaturated zone	0.3	Unitless
ρ_s	Bulk density at source	1.5	g/cm^3
ρ_u	Bulk density in unsaturated zone	1.5	g/cm^3
ρ_a	Bulk density of aquifer	1.9	g/cm^3
K_{ds}	Sorption coefficient in source	contaminant-specific	mL/g
K_{du}	Sorption coefficient in unsaturated zone	contaminant-specific	mL/g
K_{da}	Sorption coefficient in aquifer	contaminant-specific	mL/g
η	Porosity of aquifer	0.1	Unitless
T	Depth to aquifer below contamination zone	site-specific	M
α_L	Dispersivity in the direction of aquifer flow	9	M
α_T	Dispersivity perpendicular to direction of flow	4	M
Q_I	Initial contaminant mass or activity	site- and contaminant-specific	mg or Ci
$t_{1/2}$	Half-life of contaminant	contaminant-specific	Y
EWST	Equivalent well screen thickness	15	M
X	Distance from source to receptor, parallel to flow	site-specific	M
Y	Distance from source to receptor, perpendicular to flow	site-specific	M

a. Values are default Track 2 numbers unless otherwise noted.

Source length and width derivation is described in text.

The thickness of the source volume is based on predicted leached depth, as described in text.

Sorption coefficients are, in this analysis, identical for source, unsaturated, and saturated zones.

Depth to aquifer is the cumulative vadose zone interbed thickness for each site.

Distance from source to receptor is unique for each source/receptor, based on location of ten receptors at downgradient edge of this system.

Table 6-7. Modeling details for each site.

Site	UTM[a] (East, m)	UTM (North, m)	Offset[b] parallel to flow (m)	Offset perpendicular to flow (m)	CVZIT[c] (m)	Length (parallel to flow) (m)	Width (perpendicular to flow) (m)	Thickness of source (m)	Area (m^2)	Volume (m^3)	Contaminated Soil Mass (kg)
CFA-13	342910.0	4821062.0	-577.3	174.9	14.0	5.0	5.0	9.1	25.0	227.5	3.41E+05
CFA-15	342759.7	4820694.4	-209.7	24.5	13.5	0.5	0.5	7.9	0.3	2.3	3.46E+03
CFA-04	342735.2	4820484.7	0.0	0.0	14.0	150.7	45.6	5.5	6875.3	37813.2	5.67E+07
CFA-17a	343402.6	4828685.0	-8200.2	667.4	18.5	48.6	33.5	3.8	1629.9	6217.1	9.33E+06
CFA-17b	343390.0	4828718.5	-8233.7	654.9	18.5	18.3	18.1	3.8	331.1	1262.9	1.89E+06
CFA-47	343443.6	4828685.0	-8200.2	708.4	18.5	1.0	1.0	3.8	0.9	3.5	5.27E+03
CFA-07a	343547.3	4821934.0	-1449.2	812.1	46.3	2.7	2.7	3.5	7.3	25.5	3.83E+04
CFA-07b	343550.0	4821936.7	-1451.9	814.8	46.3	2.7	2.7	3.5	7.3	25.5	3.83E+04
CFA-12	342728.6	4821453.5	-968.7	-6.5	12.9	3.7	3.7	2.6	13.4	34.8	5.22E+04
CFA-08	343737.4	4821772.8	-1288.0	1002.2	47.0	305.0	61.0	9.9	18605.0	184189.5	2.76E+08
CFA-08b	344019.3	4822252.9	-1768.1	1284.1	49.0	62.4	89.2	7.6	5566.1	42302.2	6.35E+07
CFA-10	343182.5	4820914.5	-429.8	447.4	19.0	40.7	19.9	3.0	808.1	2463.1	3.69E+06
CFA-26	342821.6	4820851.5	-366.7	86.4	13.0	30.5	30.5	5.0	930.3	4651.3	6.98E+06
CFA-42	343695.9	4821661.0	-1176.2	960.7	40.9	9.1	9.1	0.2	83.6	12.7	1.91E+04
CFA-05	343660.0	4820999.0	-514.3	924.9	32.0	69.5	69.5	5.8	4829.2	27965.7	4.19E+07
CFA-05b	343590.7	4821014.8	-530.1	855.5	31.5	69.5	37.8	5.8	2626.4	15209.4	2.28E+07
CFA-52	342945.6	4821205.3	-720.5	210.5	13.5	3.5	2.4	2.9	8.4	24.4	3.65E+04
CFA-1709	342962.7	4821246.6	-761.8	227.5	13.8	2.3	2.1	2.6	4.9	12.8	n/a[d]
CFA-2	343401.4	4828673.7	-8189.0	666.2	16.5	4.1	2.4	6.6	9.9	65.1	n/a
CFA-610	342945.3	4821255.3	-770.5	210.1	13.0	3.5	2.4	2.9	8.6	25.0	n/a
CFA-658	343251.3	4821242.8	-758.0	516.1	24.0	6.3	3.7	1.7	23.0	38.5	n/a
CFA-713-4	343043.9	4821050.1	-565.3	308.7	12.3	23.2	9.1	0.8	212.4	161.8	n/a
CFA-713-5	343047.6	4821054.8	-570.1	312.4	12.3	18.6	9.1	0.8	169.7	129.3	n/a

Table 6-7. (continued).

Site	UTM[a] (East, m)	UTM (North, m)	Offset[b] parallel to flow (m)	Offset perpendicular to flow (m)	CVZIT[c] (m)	Length (parallel to flow) (m)	Width (perpendicular to flow) (m)	Thickness of source (m)	Area (m^2)	Volume (m^3)	Contaminated Soil Mass (kg)
CFA-723	342988.1	4820973.1	-488.4	253.0	12.0	5.7	3.0	2.9	17.3	50.0	n/a
CFA-726	343109.0	4821080.0	-595.2	373.9	16.0	3.9	2.7	2.0	10.6	21.0	n/a
CFA-728	343126.9	4821130.0	-645.2	391.8	17.3	3.9	2.7	2.3	10.6	24.3	n/a
CFA-729	342973.5	4821251.1	-766.4	238.3	13.0	6.5	3.0	2.3	19.7	45.0	n/a
CFA-733	342988.1	4820973.1	-488.4	253.0	12.0	5.7	3.0	2.9	17.3	50.0	n/a
CFA-734	343002.9	4821284.7	-800.0	267.8	16.0	4.1	2.4	2.3	9.9	22.6	n/a
CFA-735	342909.2	4821326.3	-841.6	174.0	14.5	3.8	2.4	2.6	9.2	23.9	n/a
CFA-741-7	342988.1	4820973.1	-488.4	253.0	10.8	23.2	9.1	0.8	212.4	161.8	n/a
CFA-745	342950.3	4821123.8	-639.0	215.1	11.0	4.2	2.7	1.7	11.5	19.3	n/a
CFA-746	342878.1	4821146.6	-661.9	142.9	10.8	2.3	2.1	3.2	4.9	15.6	n/a
CFA-747	343290.2	4821837.0	-1352.3	555.0	47.0	4.6	2.7	4.7	12.5	59.3	n/a
CFA-748-B	342961.8	4821135.0	-650.3	226.6	11.0	4.2	2.7	1.7	11.5	19.3	n/a
CFA-750	342988.1	4820973.1	-488.4	253.0	12.0	5.7	3.0	2.9	17.3	50.0	n/a
CFA-46	342836.5	4821119.3	-634.6	101.4	12.00	5.8	5.8	6.9	33.2	228.9	n/a

a. UTM = Universal Transverse Meridian north and east coordinates in meters.

b. Offset = distance in meters of the center of each site from the center of the reference site (CFA-04) parallel and perpendicular to the groundwater flow direction. A negative value parallel to groundwater flow direction indicates the site is located upgradient of CFA-04. Positive values perpendicular to groundwater flow are sites to the east of CFA-04.

c. CVZIT = cumulative vadose zone interbed thickness

d. n/a = tanks identified in the Facility Analysis of the OU 4-13 Work Plan were modeled assuming one-tank volume of product released (see Table 6-8). Contaminant inventories for these are based not on mass of contaminated soil but on mass contained in one tank volume. Tanks at sites CFA-26 and CFA-52 have petroleum inventory estimates based on this concept but also have sampling results which are used with estimates of contaminated soil mass to calculate contaminant inventories.

Table 6-8. Parameter values for contents of modeled tanks.

Tank	Modeled Contents	Kd (mL/g)	Tank Volume (L)	Total Modeled TPH Mass (mg)
CFA-713-4	TPH-gasoline	1.4	37850	2.65E+10
CFA-713-5	TPH-gasoline	1.4	30280	2.12E+10
CFA-745	TPH-gasoline	1.4	1892.5	1.32E+09
CFA-46	TPH-diesel	1.78	18927	1.61E+10
CFA-1709	TPH-diesel	1.78	946.25	8.04E+08
CFA-2	TPH-diesel	1.78	3785	3.22E+09
CFA-52	TPH-diesel	1.78	1892.5	1.61E+09
CFA-658	TPH-diesel	1.78	3785	3.22E+09
CFA-729	TPH-diesel	1.78	3785	3.22E+09
CFA-741-7	TPH-diesel	1.78	37850	3.22E+10
CFA-746	TPH-diesel	1.78	1078.725	9.17E+08
CFA-748-B	TPH-diesel	1.78	3785	3.22E+09
CFA-750	TPH-diesel	1.78	3785	3.22E+09
CFA-26	TPH-heating oil	1.98	209700	2.08E+11
CFA-610	TPH-heating oil	1.98	1892.5	1.87E+09
CFA-723	TPH-heating oil	1.98	3785	3.75E+09
CFA-726	TPH-heating oil	1.98	1892.5	1.87E+09
CFA-728	TPH-heating oil	1.98	3785	2.06E+10
CFA-733	TPH-heating oil	1.98	20817.5	3.75E+09
CFA-734	TPH-heating oil	1.98	1892.5	1.87E+09
CFA-735	TPH-heating oil	1.98	1892.5	1.87E+09
CFA-747	TPH-heating oil	1.98	3785	3.75E+09

TPH = total petroleum hydrocarbons

Table 6-9. COPC total masses or activities in soil (sources to groundwater)

Contaminant	Modeled Decay Product[a]	Half-life (yr)	Sorption Coefficient, Kd (mL/g)[b]	Total Inventory in Soil to be Transported to Groundwater (mg or Ci)
Ac-228		7.00E-04	0.00E+00	7.84E-02
	Th-228			2.87E-05
Ag-108m		1.27E+02	9.00E+01	4.80E-05
Am-241		4.32E+02	3.40E+02	3.38E-02
	Np-237			6.96E-06
Ba-133		1.05E+01	5.00E+01	4.73E-06
Bi-212		1.15E-04	1.00E+02	7.64E-02
	Pb-208[c]			4.38E-24
Bi-214		3.80E-05	1.00E+02	6.32E-02
	Pb-210			1.14E-07
Cs-137		3.02E+01	5.00E+02	7.63E+00
Eu-152		1.36E+01	0.00E+00	6.53E-05
Pb-212		1.21E-03	1.00E+02	8.03E-02
	Pb-208			4.85E-23
Pu-238		8.78E+01	2.20E+01	7.12E-04
	U-234			2.55E-07
Pu-239/240		2.41E+04	2.20E+01	1.44E-02
Ra-226		1.60E+03	1.00E+02	2.95E-01
Tl-208		5.80E-06	0.00E+00	7.32E-02
	Pb-208			2.12E-25
U-234		2.45E+05	6.00E+00	1.17E-01
U-235		7.04E+08	6.00E+00	5.93E-02
U-238		4.47E+09	6.00E+00	1.30E-01
Arsenic		n/a[d]	3.00E+00	7.49E+08
Benzo(a)anthracene		n/a	1.19E+03	3.58E+05
Benzo(b)fluoranthene		n/a	3.69E+03	1.67E+05
Benzo(g,h,i)perylene		n/a	4.74E+03	2.98E+05
Chlorodifluoromethane		n/a	1.73E-01	6.98E+05
Di-n-butylphthalate		n/a	1.02E+02	3.42E+06
Lead		n/a	1.00E+02	5.12E+09

Table 6-9. (continued).

Contaminant	Modeled Decay Product[a]	Half-life (yr)	Sorption Coefficient, Kd (mL/g)[b]	Total Inventory in Soil to be Transported to Groundwater (mg or Ci)
Mercury		n/a	1.00E+02	5.53E+09
Phenanthrene		n/a	4.23E+01	8.11E+04
Phenol		n/a	8.64E-02	2.16E+05
Tetrachloroethene		n/a	7.89E-01	9.50E+02
1,1,1-Trichloroethane		n/a	3.27E-01	2.92E+02
TPH-diesel		n/a	1.78E+00	6.77E+10
TPH-gasoline		n/a	1.40E+00	4.90E+10
TPH-heating oil		n/a	1.78E+00	2.47E+11

a. Some parent radionuclides (Ac-228, Am-241, Bi-214, and Pu-238), have relatively short half-lives and high sorption coefficients. For these radionuclides the first daughter product (Th-228, Np-237, Pb-210, and U-234, respectively) was modeled.

Daughter product inventories were obtained from the relationship of activity and half-life:

$(\text{Activity})_{\text{daughter}} = (\text{Activity})_{\text{parent}} * [(\text{half-life})_{\text{parent}}/(\text{half-life})_{\text{daughter}}]$

b. For radionuclide contaminants with extremely short half-lives (i.e., less than 1.0 yr), the COCs were assumed to decay entirely to stable products before exiting the system. These contaminants were converted from parent curies to stable product milligrams (Pb-208 for thorium series decay chain COCs and Mo-95 for Zr-95). The Pb-208 totals were added to the stable lead inventory for these sites before modeling. Mo-95 inventory was deemed insignificant for the groundwater pathway.

c. Pb-208 is a stable form of elemental lead. The short-lived parent curies were converted to milligrams of Pb-208, which was added to the total lead inventory.

d. Half-life refers to radiological decay. Here, non-radiological COCs are considered to be free of any decay-type loss mechanisms. Half-life values were taken from the EPA Health Effects Assessment Summary Tables.

n/a Not applicable

Table 6-10. Groundwater concentrations for WAG 4.

Contaminant	Modeled Decay Product[a]	100-130 Year Concentration[b] (mg/L or pCi/L)
Ac-228 (Th-228)[c]		0.00E+00
Ag-108m		0.00E+00
Am-241 (Np-237)[c]		0.00E+00
	U-233	0.00E+00
	Th-229	0.00E+00
Ba-133		0.00E+00
Bi-212 (Pb-208)[d]		n/a[d]
Bi-214 (Pb-210)[c]		0.00E+00
Cs-137		0.00E+00
Eu-152		4.79E-03
Pb-212 (Pb-208)[d]		n/a
Pu-238 (U-234)[c]		0.00E+00
	Th-230	0.00E+00
	Ra-226	0.00E+00
	Pb-210	0.00E+00
Pu-239/240		0.00E+00
	U-235	0.00E+00
	Pa-231	0.00E+00
	Ac-227	0.00E+00
Ra-226		0.00E+00
	Pb-210	0.00E+00
Tl-208 (Pb-208)[d]		n/a
U-234		0.00E+00
	Th-230	0.00E+00
	Ra-226	0.00E+00
	Pb-210	0.00E+00
U-235		0.00E+00
	Pa-231	0.00E+00
	Ac-227	0.00E+00
U-238		0.00E+00
	U-234	0.00E+00

Table 6-10. (continued).

Contaminant	Modeled Decay Product[a]	100-130 Year Concentration[b] (mg/L or pCi/L)
	Th-230	0.00E+00
	Ra-226	0.00E+00
	Pb-210	0.00E+00
Zr-95 (Mo-95)[c]		n/a[c]
1,1,1-Trichloroethane		0.00E+00
Arsenic		0.00E+00
Benzo(a)anthracene		0.00E+00
Benzo(b)fluoranthene		0.00E+00
Benzo(g,h,i)perylene		0.00E+00
Chlorodifluoromethane		1.74E-04
Di-n-butylphthalate		0.00E+00
Lead		0.00E+00
Mercury		0.00E+00
Phenanthrene		0.00E+00
Phenol		7.10E-05
Tetrachloroethene		0.00E+00
TPH-diesel[f]		0.00E+00
TPH-gasoline		0.00E+00
TPH-heating		0.00E+00

a. Some radionuclide COCs decay to significant daughter products; the daughter product ingrowth is included here.

b. The groundwater concentrations reported in this table represent the maximum predicted in a network of ten receptor aquifer wells located in a line perpendicular to the flow direction immediately downgradient of the reference site (CFA-04).

c. Radionuclide contaminants that have short half-life relative to the vadose zone transit time were modeled as their first radioactive decay product.

These include Ac-228, Am-241, Bi-214, and Pu-238 which were modeled as Th-228, Np-237, Pb-210, and U-234, respectively.

d. Some radionuclides with very short half-life (<1.0 yr) that have no significant radioactive decay products were modeled as stable decay products. Bi-212, Pb-212, Tl-208 soil inventories were converted to stable lead which was added to the total lead inventory.

e. Zr-95 is also very short-lived with no significant radioactive decay products; the inventory of Zr-95 was converted to stable Mo-95, which was found to be an insignificant soil inventory relative to the molybdenum MCL.

f. TPH = total petroleum hydrocarbon

Table 6-11. Peak groundwater concentrations for WAG 4.

Contaminant	Modeled Decay Product[a]	Peak Concentration[b] (mg/L or pCi/L)	Time (yr) at Peak Concentration
Ac-228 (Th-228)[c]		0.00E+00	n/a[d]
Ag-108m		0.00E+00	n/a[d]
Am-241 (Np-237)[c]		3.47E-05	2.64E+03
	U-233	3.31E-07	
	Th-229	2.31E-09	
Ba-133		0.00E+00	n/a[d]
Bi-212 (Pb-208)[e]		n/a[e]	n/a[e]
Bi-214 (Pb-210)[c]		0.00E+00	n/a[d]
Cs-137		0.00E+00	n/a[d]
Eu-152		4.79E-03	4.11E+01
Pb-212 (Pb-208)[e]		n/a[e]	n/a[e]
Pu-238 (U-234)[c]		2.56E-06	4.68E+03
	Th-230	5.43E-08	
	Ra-226	6.44E-07	
	Pb-210	3.24E-08	
Pu-239/240		1.06E-02	1.70E+04
	U-235	8.32E-07	
	Pa-231	1.01E-07	
	Ac-227	1.16E-08	
Ra-226		8.49E-09	2.20E+04
	Pb-210	8.61E-09	
Tl-208 (Pb-208)[e]		n/a[e]	n/a[e]
U-234		3.54E+00	1.35E+03
	Th-230	2.50E-02	
	Ra-226	6.20E-03	
	Pb-210	5.94E-03	
U-235		2.56E-01	1.35E+03
	Pa-231	5.08E-03	
	Ac-227	5.70E-04	
U-238		3.91E+00	1.35E+03
	U-234	1.49E-02	
	Th-230	5.38E-05	

Table 6-11. (continued).

Contaminant	Modeled Decay Product[a]	Peak Concentration[b] (mg/L or pCi/L)	Time (yr) at Peak Concentration
	Ra-226	9.20E-06	
	Pb-210	8.61E-06	
Zr-95 (Mo-95)[f]		n/a[f]	n/a[f]
1,1,1-Trichloroethane		6.22E-08	1.20E+02
Arsenic		4.40E-02	6.96E+02
Benzo(a)anthracene		1.23E-08	2.88E+05
Benzo(b)fluoranthene		1.85E-09	8.92E+05
Benzo(g,h,i)perylene		1.83E-09	1.15E+06
Chlorodifluoromethane		1.74E-04	7.79E+01
Di-n-butylphthalate		2.98E-06	2.22E+04
Lead		1.33E-03	5.02E+04
Mercury		1.03E-02	2.18E+04
Phenanthrene		3.83E-08	2.42E+04
Phenol		7.10E-05	5.91E+01
Tetrachloroethene		1.05E-07	2.29E+02
TPH-diesel[g]		3.74E+00	4.27E+02
TPH-gasoline		6.89E+00	3.30E+02
TPH-heating		7.95E+00	4.64E+02

a. Some radionuclide COCs decay to significant daughter products; the daughter product ingrowth is included here. For this analysis, daughter products are assumed to travel at the same rate as the parent.

b. The groundwater concentrations reported in this table represent the maximum predicted in a network of ten receptor aquifer wells located in a line perpendicular to the flow direction immediately downgradient of the reference site (CFA-04).

c. Radionuclide contaminants that have short half-life relative to the vadose zone transit time were modeled as their first radioactive decay product. These include Ac-228, Am-241, Bi-214, and Pu-238 which were modeled as Th-228, Np-237, Pb-210, and U-234, respectively.

d. Some radioactive contaminants decay to stable products before reaching any receptor well locations.

e. Some radionuclides with very short half-life (<1.0 yr) that have no significant radioactive decay products were modeled as stable decay products. Bi-212, Pb-212, Tl-208 soil inventories were converted to stable lead which was added to the total lead inventory (see results for lead).

f. Zr-95 is also very short-lived with no significant radioactive decay products; the inventory of Zr-95 was converted to stable Mo-95, which was found to be an insignificant soil inventory relative to the molybdenum MCL.

g. TPH = total petroleum hydrocarbon

Assumptions

The contaminant concentrations shown in Tables 6-10 and 6-11 are expected to overestimate the true aquifer concentrations that will be produced by infiltration of contaminants at WAG 4. Because of the complicated flow and transport of contaminants in groundwater, the uncertainty about potential contaminant concentrations associated with the groundwater pathway exposure routes is greater than the uncertainty associated with any other exposure pathway in this BRA. To compensate for this relatively large uncertainty, assumptions are used throughout the groundwater pathway analysis that will cause calculated COPC concentrations to be higher than would be expected. Some of the conservative assumptions that are used in the GWSCREEN analysis are as follows:

- For the purposes of groundwater modeling, infiltration of precipitation at WAG 4 is assumed to be spatially uniform. Increase in the value of this parameter at one site or another due to accumulation of runoff from other areas and, conversely, decrease in the value due to drainage of runoff are not considered. The INEEL Track 2 default value of 0.10 cm/yr is considered an acceptable value for this parameter.

- For the purposes of this modeling, the unsaturated zone is defined as the zone between the base of the source volume and the top of the aquifer. The unsaturated zone is assumed to be homogeneous, isotropic, porous media with constant, unidirectional flow in the vertical (downward) direction. Due to the complexities and inherent uncertainties in modeling unsaturated flow and transport, a simplified plug-flow model is used that does not account for dispersion in the unsaturated zone. In the plug flow model, non-sorbing contaminants move with the vertical velocity of the water. This velocity is calculated assuming the unit hydraulic gradient (gravity drainage) condition.

  For most contaminants, the plug flow model is a conservative assumption since the peak flux to the aquifer exceeds the peak flux when dispersion is considered. Although plug flow leads to higher predicted contaminant concentrations in the aquifer, predictions of contaminant travel time to the aquifer are lower when vadose zone dispersion is included. Recent versions of GWSCREEN incorporate vertical dispersion in the unsaturated zone; however, values for the unsaturated zone vertical dispersivity are currently gross estimates only.

- In the unsaturated zone, water and contaminants are assumed to flow through basalt sequences very quickly relative to flow through sediment. As a result, the accepted Track 2 method is to ignore the vertical thickness of basalt in the vadose zone. For modeling purposes, unsaturated flow is assumed to occur through surficial and interbed sediments only.

  The accepted Track 2 method for estimating the vadose zone thickness, devoid of basalt, is to sum the known surficial sediment thickness and interbed thicknesses between the base of the source and the top of the aquifer or, when little subsurface data are available, to use one-tenth the overall depth to the aquifer. For the WAG 4 groundwater modeling, available lithological data from well logs were analyzed to provide site-specific cumulative interbed thicknesses.

  For non-decaying contaminants, the only effect variations in the total unsaturated zone sediment thickness have are on the unsaturated transit time and, hence, contaminant arrival times at the receptor. All of the non-decaying contaminant will eventually reach the aquifer

regardless of the unsaturated sediment thickness. The sensitivity of groundwater modeling predictions to changes in this parameter are investigated in Appendix F.

- For radiological contaminants, the mass of contaminant reaching the aquifer and, hence, aquifer concentrations at the receptor are affected by changes in the unsaturated zone sediment thickness. Decay of the radiological contaminant in the unsaturated zone will be enhanced by greater sediment thicknesses. Large adsorption coefficients for some radiological contaminants will further enhance this vadose zone decay.

- All COPC mass contained in surface soils at WAG 4 is assumed to contribute to groundwater contamination. No credit is taken in the modeling for loss of COPC mass caused by mechanisms such as wind erosion, surface water erosion, contaminant uptake into plants, biodegradation, etc. The only contaminant loss mechanism that is considered in the groundwater pathway evaluation is radioactive decay.

Two other assumptions included in the groundwater analysis, but not limited to the GWSCREEN modeling, are as follows:

- The groundwater receptor is assumed to take all drinking water for 30 years from one of ten receptor well locations along the downgradient edge (with respect to groundwater flow) of WAG 4.

- All contaminants are assumed to be uniformly distributed within the groundwater modeling source volume.

Model Selection

The groundwater modeling performed for the WAG 4 RI/FS is based on the solution algorithms of the semi-analytical model GWSCREEN (Rood 1994). GWSCREEN is used widely at the INEEL for assessment of groundwater pathway impacts from surface or buried contamination. The algorithms of one of the latest versions of the code, version 2.4a, are incorporated in a pre- and post-processing user-interface called GWMENU. This interface allows integration of input and output from the model for easier computation of cumulative impacts of more than one site at a common receptor.

Although formal documentation for GWMENU is not available, the interface's similarity to GWSCREEN version 2.4a is confirmed as part of the groundwater modeling sensitivity analysis included in Appendix F. Runs were made using the GWMENU program and compared with runs by GWSCREEN version 2.4a using the same input parameters.

The GWSCREEN family of codes was designed to perform groundwater pathway screening calculations for the Track 1 and 2 process. It is an appropriate model to use when site characterization data are lacking and little would be gained by the use of more complex models. Other more sophisticated models were reviewed but were not selected to perform groundwater pathway calculations because:

- The probability of potential risks exceeding acceptable criteria was low and did not warrant the use of a more sophisticated model at this time in the process,

- Additional field characterization data would likely need to be obtained to fully utilize the capabilities of a more complex model, and

- The results of this analysis will indicate what operable units, if any, require further attention; the use of a more complex model at such time may be justified.

Source Areas

Source areas were modeled individually instead of modeling a single WAG 4 composite site. Each release site within WAG 4 was represented by rectangular areas that were oriented parallel to north-south or east-west lines. Dimensions used in the model, as described in Table 6-7, are modified from actual site dimensions to account for irregular geometry and source orientation. The total volumes of each modeled source, however, were made to approximate the actual estimated contaminated volumes (see Table 6-7). A total of 13 sites, retained following the supplemental contaminant screening performed in this report (Section 6.2..2.2) were modeled.

Some of the retained sites required several rectangular areas (elements) to represent the source. These include CFA-05, CFA-07, CFA-17. CFA-05, the motor pool pond, includes the pond as well as a ditch (two elements); CFA-07 includes two french drains (two elements); CFA-17 includes a rectangular fire training pad and a leach pit (two elements). CFA-08 contains two elements also; one representing the sewage treatment plant, the other represents the drain field.

In addition, the modeling included 22 aboveground and underground fuel storage tanks identified in the facility analysis portion of the OU 4-13 Work Plan. Simplifying assumptions were used to incorporate these tanks for which little data are available. These include assuming only one tank volume of fuel product leaked from each tank into the subsurface during the life of the tank. This assumption is reasonably conservative because operators of the tank systems would have probably noticed larger leaks.

Each retained site was located according to its physical geographic location within the CFA facility. Two sites are located outside of CFA; although CFA-17 and CFA-47 are retained in the OU 4-13 effort, they are located on Lincoln Avenue approximately 6 km (4 mi) north of CFA. Table 6-7 presents the Universal Transverse Meridian (UTM) north and east coordinates for each site.

A receptor grid was overlain on the source areas such that contributions to individual contaminant groundwater concentrations from all retained sites could be calculated at each receptor node. CFA-04 served as the most downgradient site, with respect to groundwater flow, and as such served as the model reference site. With the exception of CFA-12, CFA-04 is also the western-most site that was modeled. Contaminant groundwater concentrations were determined for each of ten receptor locations spread across an east-west line at the downgradient edge of CFA-04 that extends from 200 m (658 ft) west of the center of CFA-04 to 1200 m (3,947 ft) east of CFA-04.

A common receptor grid allows groundwater concentrations of contaminants common to two or more sites to be summed for determining cumulative impacts. The concentrations reported in Tables 6-10 and 6-11 are these cumulative groundwater concentrations. More detail on the design of the groundwater model including the receptor network is provided with the sensitivity analysis and output files in Appendix F.

The spacing between receptors varies; the western most receptor is 200 m (658 ft) from the center of CFA-04, the next five receptor locations are spaced 100 m (329 ft) apart, and the final four receptors to the east spaced at 200 m (658 ft). This was done because the majority of sites modeled for the groundwater pathway are located within 500 m (1,645 ft) east of CFA-04. The distance from the center of CFA-04 to the center of each area source was computed (listed in columns 4 and 5 of Table 6-7) which provided a means to relate the receptor grid to all source areas in the model domain. Additionally, a

receptor well was located at the downgradient edge of each source. The results for the source edge are not reported in this section but can be found in the appendix containing model output (Appendix F).

Each source area was modeled as a surficial or buried source as described in the GWSCREEN user's manual (Rood 1994). These sources are modeled in GWSCREEN using the assumption of homogeneously mixed contamination within the source volume and with natural infiltration of precipitation as the only downward driver for contaminant migration. Steady state infiltration under unit hydraulic gradient conditions are assumed. Contaminants area assumed to be in solid and aqueous phase equilibrium and equilibrium concentrations are described by the linear sorption coefficient, K_d. Leaching from the source volume to the underlying strata is assumed to be a first-order process where the fraction leached is constant and the mass flux is proportional to the amount of contaminant present in the source volume. Radioactive decay is considered but all other loss mechanisms, including biodegradation of petroleum products, are ignored.

Unsaturated Zone

The Track 1 and 2 groundwater modeling process typically only considers water travel time through sedimentary interbeds and assumes the transit time through fractured basalt is relatively instantaneous. The Large Scale Infiltration Test, performed at the INEEL in 1995, came to a similar conclusion that transit time in the unsaturated zone is controlled by the hydraulic properties of the sedimentary interbeds and not the fractured basalt. This approach has been incorporated into this analysis. Interbed thickness is known to vary across CFA. Site-specific well logs have been used to delineate the interbed thickness below the retained sites. The advantage to the conceptual model employed in this analysis is that each source area may be assigned an interbed thickness that coincides with the actual estimated interbed thickness underlying the source. Using a single composite source does not allow for this refinement. The sensitivity of predicted groundwater concentrations to changes in the unsaturated zone sediment thickness are analyzed in Appendix F.

The unsaturated zone in GWSCREEN is modeled using a plug flow model. Dispersion and diffusion are ignored in the vadose zone; only radioactive decay is allowed to reduce contaminant concentrations in the leachate. Unit gradient conditions are assumed throughout the unsaturated zone. Contaminant travel times are governed by the water infiltration rate, contaminant-specific sorptive properties, and the hydraulic properties of the interbeds.

Saturated Zone

The saturated zone (Snake River Plain Aquifer) is modeled as a homogeneous isotropic aquifer of infinite lateral extent and finite thickness. No sources or sinks are considered and a steady state uniform flow field is assumed. Contaminants enter the aquifer in an area defined by the length and width of the source area and disperse both horizontally and vertically as they are transported downgradient. Equilibrium sorption reactions described by the K_d value are included. Contaminant concentrations are evaluated by averaging the concentration in the first 15 m (49 ft) of the aquifer (measured from the water table, the top surface of the aquifer). The 15 m (49 ft) averaging concentration depth was chosen based on the default Track 2 length of well screen (DOE/ID 1994). Contaminants are allowed to disperse completely in this effective aquifer thickness as they move downgradient.

Groundwater flow direction underneath CFA is approximately south to southwest. To simplify the GWSCREEN calculations, the flow direction is assumed to be directly south. This assumption may be nonconservative for some sites, overly conservative for others, but the overall effect is expected to be minimal. Gradients in the SRPA underneath CFA are relatively shallow and the actual flow direction is not precisely known. For sites with significantly different length and width dimensions, groundwater

flow direction will influence the estimated maximum concentration. Sources such as CFA-05 may generate higher groundwater concentrations when the groundwater flow is oriented in a more western direction. Groundwater concentration results were calculated primarily for groundwater flow in the north-south direction because:

- The estimated flow path is nearer to the north-south direction compared to east-west.
- Elongated source areas such as CFA-05 are actually more complex, containing several points at which source orientation changes with respect to groundwater flow.
- Groundwater flow orientation has little impact on maximum concentrations predicted for sources with approximately equal length and width.
- A change in groundwater flow direction will increase predicted concentrations from some source areas while decreasing concentrations from others.

To quantify the uncertainty associated with the groundwater flow direction and to provide the sensitivity of predicted groundwater concentrations to changes in the flow direction, the groundwater modeling sensitivity analysis in Appendix F includes analysis of flow direction. The model was rerun with different flow directions and the results compared with the base case (flow directly south) presented here."

Groundwater Transport Parameters

In this section, the input parameter values for the GWSCREEN model for each retained, modeled site source element are described. Parameter values are provided for the source, unsaturated, and saturated zone. Contaminant sorption coefficients values (K_d) used in this analysis and listed in Table 6-9 are the same as those used in the human health risk assessment and are shown by contaminant in Appendix F.

Source Area Parameters

Three input parameters shown in Table 6-7 (length of source parallel to groundwater flow, width of source perpendicular to groundwater flow, and thickness of source) are based on a variety of dimensional estimating methods. These include known estimates based on assumed contaminant release volumes, soil porosity, and depth to basalt; known extents of soil contamination, where available; and estimates based on the square root of a known surface contaminated area. Thickness of contamination was based on sampling results; however, vertical extent of contamination for the groundwater pathway includes an estimated depth to which contamination is suspected to have leached. Retained site source areas were oriented such that their sides were parallel to north-south or east-west trending lines. Tanks were oriented such that their longest dimension is parallel to the flow direction; extent of contamination was based on one tank volume released, residual soil porosity, and depth to basalt. Other tank input parameters are shown in Table 6-8.

Surface soil hydraulic properties were obtained from the GWSCREEN user's manual (Rood 1994). Default Track 1 and 2 moisture contents and an annual infiltration rate of 10 cm/y were used as input. For contaminated source zones, their respective infiltration rates provide the driving force for the transport of contaminant from beneath these surface features, through the remaining vadose zone, and into the aquifer. These infiltration rates are assumed to result from natural sources only. Natural sources of

infiltrating water include precipitation as either rain that falls directly on to the contaminated zone surface or snowmelt from snow that falls directly on the surface feature.

Natural infiltrating water can also include rainfall or snowmelt from other areas of the site that may flow as runoff and collect on the surface of contaminated zones. The natural background infiltration rate for these sites is a function of precipitation, evapotranspiration, and other factors. The widely accepted infiltration rate for the INEEL is taken from Track 2 guidance (DOE 1994) as 0.1 m/yr. This rate is assumed for all contaminated source zones modeled for the groundwater pathway. Potential runoff from other parts of the facility that may collect above contaminated zones is ignored in this analysis.

Unsaturated Zone Parameters

Interbed thickness is known to vary considerably across the INEEL. Well logs have been used to delineate thickness at different locations within WAG 4. As noted On Table 6-5, data available for associated delineation of the interbeds is limited. Isopleth contours of interbed thickness were generated using a Kriging interpolation routine that is part of the Surfer® software package (Golden Software Inc. 1996). All source areas are underlain by sedimentary interbeds of varying thickness. The sum thickness of the interbeds beneath a contaminated site and above the top of the aquifer is referred to here as the cumulative vadose zone interbed thickness (CVZIT). The CVZIT values for CFA area aquifer wells, used to prepare the kriged contours, are listed in Table 6-5. CVZIT values estimated for each modeled site are listed in Table 6-7.

The effects of varying the CVZIT parameter are analyzed in the sensitivity analysis of Appendix F. The input parameter values and modeled results presented in this section serve as the base case for the Appendix F analysis. The sensitivity analysis includes examination of effects of minimum and maximum unsaturated zone sediment thicknesses for each modeled site. It also includes the effects of discontinuous interbeds at WAG 4 and in areas downgradient of WAG 4.

Saturated Zone Parameters

Saturated zone parameters were obtained from the Track 1 and 2 guidance manuals (DOE 1992 and 1994) and are listed in Table 6-6. Values for effective porosity, dispersivity, and well screen thickness were obtained from the Track 1 and 2 guidance manuals. A pore velocity (average linear velocity) for the Snake River Plain Aquifer near CFA of 570 m/y is a default Track 2 parameter. The effective aquifer thickness was assigned a default value of 76 m.

The aquifer model used in this analysis included dispersion in three directions. Therefore, a vertical dispersivity value was required. Typically, the vertical dispersivity is assumed to be the same as the transverse dispersivity. In order to provide some conservatism to the calculation, vertical dispersivity was assigned a value 10 times less than the transverse dispersivity 0.4 m (1.3 ft). Contaminants were allowed to disperse into the effective thickness of the aquifer (76 m). Output concentrations were based on the average aquifer concentration in the first 15 m (49 ft) of aquifer (the well screen thickness) measured from the surface. Near the source, little vertical mixing occurs and calculated concentrations are similar to those calculated using a vertically averaged model and 15 m (49 ft) mixing thickness. Note, this is the same approach used in the Track 1 and 2 process. Farther away from the source, the contaminant plume disperses beyond the 15 m (49 ft) well screen depth. Therefore, contaminant concentrations are lower compared to those calculated assuming a constant 15 m (49 ft) mixing thickness.

Maximum concentrations in the aquifer were calculated for each contaminant; both the peak concentrations, occurring at any time, and concentrations occurring during 100–130 years from present

were obtained. The values presented in Table 6-10 and Table 6-11 are the highest 30-yr average concentrations of the set of ten receptor locations. Contaminant concentrations at each receptor node were calculated separately for each source and then summed across all sources. Therefore, concentrations for a specific contaminant at each receptor node represents contributions from all sources considered in the assessment.

Appendix F contains the GWSCREEN output files for the peak concentration simulations prepared for each COPC. Table 6-10 and 6-11 summarize the predicted groundwater concentrations.

It should be noted that short-lived radionuclides with significant (long-lived) decay products and high sorption coefficients were modeled as their first long-lived daughter product. This assumes the parent nuclide inventory will transform to the daughter product while still bound in the vadose zone. Am-241 and Pu-238 are two such parent radionuclide contaminants at WAG 4; they were modeled with GWSCREEN as Np-237 and U-234, respectively. Additionally, Ac-228 and Bi-214 were modeled as Th-228 and Pb-210, respectively.

Other short-lived radionuclide contaminants with no significant long-lived daughter products were modeled as their final stable decay product. Bi-212, Pb-212, and Tl-208 were all converted from curies of each to milligrams of stable Pb-208. These inventories of Pb-208 were then added to the total lead inventory at this site (CFA-05). The inventory of Zr-95, present at CFA-13, was converted from curies of Zr-95 (6E-06 Ci) to milligrams of stable Mo-95, which has a MCL of 0.18 mg/L. The resulting inventory of Mo-95 (2E-25 mg) is too insignificant to warrant further groundwater modeling.

Finally, the individual BTEX (benzene, toluene, ethylbenzene, and xylene) compounds, typically risk-drivers for petroleum contaminated sites, were not modeled separately for petroleum contamination sites at WAG 4. The petroleum release sites were modeled assuming the release of one-tank volume of petroleum product. The relative concentrations of BTEX compounds in petroleum products vary considerably and are a function of the proprietary mix of chemicals used by petroleum vendors. As a result, the petroleum sites were modeled as TPH of either gasoline, diesel, or heating oil.

6.3.3.3.1 Groundwater Ingestion—The groundwater pathway is evaluated on a WAG-wide basis. As with the soil and air pathways, groundwater pathway risks and HQs are calculated for the 100 year residential scenario.

6.3.3.3.2 Dermal Absorption From Groundwater—Exposures to COPCs through dermal absorption of groundwater are controlled by the chemical-specific permeability coefficient of water through skin (K_p^w). According to EPA guidance (EPA 1992c), if the permeability coefficient for a given COPC is less than 0.1 cm/hour, then the dermal absorption from groundwater exposure route results in potential risks that are less than potential risks via the groundwater ingestion exposure route for that COPC. In the BRA, the default permeability coefficient used for inorganic COPCs is 1E–03 cm/hour, and the permeability coefficients for organic COPCs are estimated using the following equation:

$$\text{Log } K_p^w = -2.72 + 0.71 \text{ Log } K_{ow} - 0.0061 \text{ MW} \qquad (6\text{-}13)$$

where

Kow = octanol/water partition coefficient (unitless)

MW = molecular weight (g/mol).

Permeability coefficients for WAG 4 COPCs are shown in Table D-14. Because many of the organics have permeability coefficients that are greater than the screening level of 0.1 cm/hr, the dermal absorption from the groundwater exposure route is quantitatively evaluated in the BRA. Contaminant intakes for this exposure route are calculated using the equations shown in Section 6.3.4.

6.3.3.3.3 Inhalation of Groundwater VOCs. Exposures caused by the inhalation of water vapors from indoor water use are calculated based on experimental data derived from a study of household water contaminants (Andelman 1990). This study derived a volatilization constant that defines the relationship between the concentration of a contaminant in household water and the average concentration of the volatilized contaminant in air. All uses of household water were considered (e.g., showering, laundering, and dish washing), and certain reasonable assumptions were made in deriving a volatilization fraction. For example, the study included assumptions about water usage for a family of four, the volume of the dwelling, and the air exchange rate. Furthermore, the study assumed that the average transfer efficiency weighted by the type of water use is 50% (i.e., half of the concentration of each chemical in water will be transferred into air by all types of water uses).

Indoor water use analysis, a central tendency value [6.50E–02 mg/m^3 air per mg/L water (Andelman 1990)] for the volatilization fraction of a COPC is used in the BRA to develop estimates of COPC airborne concentrations. The airborne concentrations are calculated by multiplying the central tendency value by the COPC groundwater concentrations shown in Table D-45. These concentrations are then used to develop contaminant intake estimates using the equations shown in Section 6.3.4. The estimates of COPC airborne concentrations from indoor water use calculated for the BRA are shown in Table D-22.

6.3.4 Estimation of Contaminant Intakes

The general equation that is used to calculate intakes for most of the BRA exposure routes is as follows (EPA 1989a):

$$Intake = \frac{C \times IR \times EF \times ED}{BW \times AT} \qquad (6\text{-}14)$$

where

Intake = contaminant intake (mg/kg-day)

C = concentration of a given contaminant in a contaminated medium (soil, air, water, etc.) (mg/kg, mg/m^3, mg/L, etc.)

IR = ingestion rate of the contaminated medium (mg/day, m^3/day, L/day, etc.)

EF = exposure frequency (day/year)

ED = exposure duration (year)

BW = body weight (kg)

AT = averaging time (day).

The above equation applies to all exposure routes except exposure to external radiation. For the external radiation exposure route, intakes are calculated using the following general equation:

$$\text{Intake} = C \times ET \times EF \times ED \times CF \quad (6\text{-}15)$$

where

Intake = radiation intake (pCi-year/g)

C = radionuclide concentration in soil (pCi/g)

ET = exposure time (hour/day)

EF = exposure frequency (day/year)

ED = exposure duration (year)

CF = conversion factor (1.14E–04 year/hour).

The exposure assumptions used to calculate intake factors for the occupational and residential exposure scenarios are summarized in Table D-23. Exposure pathway-specific intake factor equations are presented in Tables D-24 through D-35. Tables D-36 through D-39 present the calculated intake factors for the current and future occupational exposure scenario. Tables D-40 and D-41 present the calculated intake factors for the future residential scenario.

6.4 Toxicity Assessment

This section provides the toxicity constants that will be used for risk characterization purposes and summarizes toxicological information for the WAG 4 radioactive and nonradioactive COPCs. For this assessment, and consistent with *EPA's RAGS* (EPA 1989a), the toxicity information is summarized for two broad categories of potential effects: noncarcinogenic and carcinogenic effects. These two categories are selected because of the slightly differing methodologies for estimating potential health risks associated with exposures to carcinogens and noncarcinogens.

The toxicity constants used in the BRA are obtained from several sources. The primary source of information is the EPA's Integrated Risk Information System (IRIS). IRIS contains only those toxicity constants that have been verified by EPA's Reference Dose or Carcinogen Risk Assessment Verification Endeavor Work Groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPA's HEAST (EPA 1995a) are used. The toxicity constant tables are published annually and updated approximately twice per year. HEAST contains a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, agency-wide information (EPA 1995a).

6.4.1 Toxicity Assessment for Carcinogenic Effects

Potential carcinogenic risks are expressed as an estimated probability that an individual might develop cancer from lifetime exposure. This probability is based on projected intakes and chemical-specific dose-response data called cancer SFs. Cancer SFs and the estimated daily intake of a compound, averaged over a lifetime of exposure, are used to estimate the incremental risk that an

individual exposed to that compound may develop cancer. This estimate is derived using the following equation:

$$\text{Risk} = \text{Intake} \times \text{SF} \qquad (6\text{-}16)$$

where

Risk = Carcinogenic risk (unitless)

Intake = Chemical intake (mg/kg-day or pCi)

SF = Slope factor ($[\text{mg/kg-day}]^{-1}$ or $[\text{pCi}]^{-1}$).

There are two classes of potential carcinogens identified at WAG 4 release sites: chemical carcinogens and radionuclides. These two classes of carcinogens are discussed separately in the following subsections.

6.4.1.1 Toxicity Assessment for Chemical Carcinogens. Evidence of chemical carcinogenicity originates primarily from two sources: (1) lifetime studies with laboratory animals and (2) human (epidemiological) studies. For most chemical carcinogens, animal data from laboratory experiments represent the primary basis for the extrapolation. Major assumptions arise from the necessity of extrapolating experimental results: across species (i.e., from laboratory animals to humans); from high-dose regions (i.e., levels to which laboratory animals are exposed) to low-dose regions (i.e., levels to which humans are likely to be exposed in the environment); and across routes of administration (i.e., inhalation versus ingestion). Federal regulatory agencies have traditionally estimated human cancer risks associated with exposure to chemical carcinogens on the administered-dose basis according to the following approach:

- The relationship between the administered dose and the incidence of cancer in animals is based on experimental animal bioassay results.
- The relationship between the administered dose and the incidence of cancer in the low-dose range is based on mathematical models.
- The dose-response relationship is assumed to be the same for both humans and animals, if the administered dose is measured in the proper units.

Effects from exposure to high (i.e., administered) doses are based on experimental animal bioassay results, while effects associated with exposure to low doses of a chemical are generally estimated from mathematical models.

For chemical carcinogens, EPA assumes a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and tumor induction. This mechanism for carcinogenesis is referred to as stochastic, which means that there is theoretically no level of exposure to a given chemical that does not pose a small, but finite, probability of generating a carcinogenic response.

Because risk at low exposure levels cannot be measured directly either in laboratory animals or human epidemiology studies, various mathematical models have been proposed to extrapolate from high to low doses (i.e., to estimate the dose-response relationship at low doses). The three most frequently used models are (1) the one-hit model, (2) the log-probit model, and (3) the multistage model. The

one-hit model is based on the premise that a single molecule of a chemical can be the single event that precipitates tumor induction (Cornfield 1977). In other words, there is some finite response associated with any exposure. The log-probit model assumes that a response is normally distributed with the logarithm of the dose (Mantel et al. 1971). This theory seems to have little scientific basis, although some physiological parameters are lognormally distributed. This model usually yields much lower potency estimates because of the implied threshold at lower doses.

Currently, regulatory decisions are based on the output of the linearized multistage model (EPA 1989a). The basis of the linearized multistage model is that multiple events (versus the single-event paradigm of the one-hit model) may be needed to yield tumor induction. The linearized multistage model reflects the biological variability in tumor frequencies observed in animals or human studies (Crump et al. 1977). The dose-response relationship predicted by this model at low doses is essentially linear. Use of this model provides dose-response estimates intermediate between the one-hit and the log-probit models. It should be noted that the SFs calculated for nonradiological carcinogens using the multistage model represent the 95th percentile UCL on the probability of a carcinogenic response. Consequently, risk estimates based on these SFs are conservative estimates representing upper-bound estimates of risk where there is only a 5% probability that the actual risk is greater than the estimated risk.

Most models produce quantitatively similar results in the range of observable data, but yield estimates that can vary by three or four orders of magnitude at lower doses. Animal bioassay data are simply not adequate to determine whether any of the competing models are better than the others. Moreover, there is no evidence to indicate that the precision of low-dose risk estimates increases through the use of more sophisticated models. Thus, if a carcinogenic response occurs at the exposure level studied, it is assumed that a similar response will occur at all lower doses, unless evidence to the contrary exists.

Uncertainties in the toxicity assessment for chemical carcinogens are dealt with by classifying each chemical into one of several groups, according to the weight of evidence from epidemiological studies and animal studies, as follows (EPA, 1989a):

- Group A—Human carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B—Probable human carcinogen (B1–limited evidence of carcinogenicity in humans; B2–sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C—Possible human carcinogen (limited evidence of carcinogenicity in the animals and inadequate or lack of human data)
- Group D—Not classifiable as to human carcinogenicity (inadequate or no evidence)
- Group E—Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

Table D-42 provides the SFs, in $(mg/kg/day)^{-1}$, and the weight-of-evidence for each WAG 4 COPC.

To obtain an estimate of total carcinogenic risk resulting from modeled exposures to carcinogens at the site, cancer risks are summed across all exposure routes for all carcinogens. Cancer risks from

exposure to multiple carcinogens across multiple pathways are assumed to be additive, based on EPA carcinogen risk assessment guidelines (EPA 1986).

6.4.1.2 Toxicity Assessment for Radionuclides. An extensive body of literature exists that describes the health effects of radionuclides on humans and animals. Intensive research by national and international commissions has resulted in the establishment of widely accepted limits to which workers and the public may be exposed without clinically detectable effects. This literature has resulted in EPA classifying all radionuclides as Group A carcinogens because radionuclides emit ionizing radiation, which, at high doses, has been associated with increased cancer incidence in humans. Human epidemiological data collected from the survivors of the Hiroshima and Nagasaki bomb attacks form the basis for the most recent extrapolation put forth by the National Academy of Science (NAS 1980). Conversely, for most nonradiological carcinogens, animal data from laboratory studies represent the primary basis for the extrapolation.

Another fundamental difference between the assessment of potential toxicity associated with exposure to radionuclide and nonradionuclide carcinogens is that SFs for radionuclides are typically best estimates (mean or median values rather than upper 95th-percentile values). Furthermore, in the past, risk factors for radionuclides have generally been based on fatalities (i.e., the number of people who actually died from cancer), while SFs for nonradiological carcinogens are based on incidence (i.e., the number of people who developed cancer). Finally, the SFs for radionuclides are expressed in different units, i.e., risk per $(pCi)^{-1}$ rather than $(mg/kg/day)^{-1}$.

Table D-42 lists SFs for all radionuclides identified at WAG 4 release sites. These nonthreshold SFs account for the following: the amount of radionuclide transported into the bloodstream, the decay of radioactive progeny within the body, the distribution and retention of the radionuclide and its progeny (if any) in the body, the radiation dose delivered to specific organs and tissues, and the age and sex of the exposed individuals (EPA 1995).

6.4.2 Toxicity Assessment for Noncarcinogenic Effects

Potential noncarcinogenic effects are evaluated by comparing daily intakes with chronic RfDs developed by the EPA. This section provides a definition of an RfD and discusses how it is applied in the OU 4-13 BRA. Table D-42 provides the RfD values for each of the COPCs identified at WAG 4 release sites.

A chronic RfD is an estimate of the daily exposure that can be incurred during a lifetime, without an appreciable risk of a noncancer effect being incurred in human populations, including sensitive subgroups (EPA 1989a). The RfD is based on the assumption that thresholds exist for noncarcinogenic toxic effects (e.g., liver or kidney damage). It is a benchmark dose operationally derived by the application of one or more order-of-magnitude uncertainty factors to doses thought to represent a lowest or no-observed-adverse-effect level (NOAEL) in humans. Thus, there should be no adverse effects associated with chronic daily intakes below the RfD value. Conversely, if chronic daily intakes exceed this threshold level, there is a potential that some adverse noncarcinogenic health effects might be observed in exposed individuals.

RfDs or SFs have not been developed by the EPA for the dermal exposure route. In the absence of these factors, the common practice has been to use the available toxicity measures for the oral route of exposure. This approach has been adopted in the BRA.

In evaluating the dermal pathway, the EPA recommends expressing chemical intake as absorbed dose and adjusting the oral toxicity measures to reflect absorbed dose (EPA 1989a). In deriving such

values, consistency is required between the type of dose that forms the basis of the oral toxicity and the type of dose that will be calculated by the dermal exposure models. Specifically, a distinction must be made between an administered dose or intake (i.e., the amount of chemical taken into the body) and the absorbed dose (i.e., the amount of chemical that crosses the body membranes and enters the bloodstream). Most of the toxicity measures available from the EPA are expressed as administered dose (i.e., intake) rather than dose at the tissue level (i.e., absorbed dose). The adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies or on the oral absorption efficiency in the species on which the toxicity measures are based. EPA notes that exposure estimates for absorption efficiency should not be adjusted if the toxicity values are based on administered doses (EPA 1989a).

For risk characterization purposes, the potential health effects of chronic exposure to noncarcinogenic compounds are assessed by calculating an HQ for each COPC. An HQ will be derived by dividing the estimated daily intake by a chemical-specific RfD as shown in the following equation:

$$HQ = Intake / RfD \quad (6\text{-}17)$$

where

HQ = Hazard quotient (unitless)

Intake = Chemical intake (mg/kg-day)

RfD = Reference dose (mg/kg-day).

An HQ greater than 1.0 indicates that exposure to a given chemical (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure to noncarcinogenic compounds does not increase linearly as HQ values exceed 1.0. In other words, HQ values do not represent a probability or a percentage. For example, an HQ of 10 does not indicate that adverse health effects are 10 times more likely to occur than an HQ value of 1.0. All one can conclude is that HQ values greater than 1.0 indicate that noncarcinogenic health impacts are possible and that the higher the HQ value, the greater the concern about potential adverse health effects.

Consistent with RAGS, chemical-specific HQs are summed across exposure routes to calculate a HI for each COPC. Individual pathway HI values are then summed to determine a cumulative HI value for all exposure pathways and COPCs at each release site. This approach may result in a situation where a total HI value for a given release site may exceed 1.0 even though none of the chemical-specific HQ values at the release site exceed 1.0.

6.4.3 Toxicity Profiles

The following subsections present general and chemical-specific information on health effects relating to the COPCs evaluated in the BRA. All information presented in these subsections is from IRIS (EPA 1997b) unless otherwise specified. Chemical-specific toxicity values for each COPC discussed in these subsections are presented in Table D-4.

6.4.3.1 Arsenic. (CAS No. 7440-38-2; As; Mol. wt. = 75 g/mol; WoE = A; Oral SF = 1.5 mg/kg-day, oral RfD = 0.00003 mg/kg-day, inhalation SF = 50 mg/kg/day). Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer; lung cancer results from

inhalation. Insufficient data exist to determine carcinogenic effects in animals. Chronic exposure, either by ingestion or inhalation, is marked by malaise and fatigue. Changes in the skin include hyperkeratosis. Anemia, neuropathy, liver injury, and "blackfoot disease" can also result from chronic exposure.

Acute exposure to arsenic causes severe throat irritation, gastrointestinal disturbance, and muscle spasms. This is followed by vertigo, delirium, and coma. Facial edema may also be evident. Sensory loss and hematopoietic symptoms associated with acute exposure are usually reversible. The oral NOAEL and LOAEL are 0.0008 mg/kg-day and 0.014 mg/kg-day, respectively with critical effects of hyperpigmentation, keratosis, and possible vascular complications. The uncertainty factor and modifying factor for the NOAEL are 3 and 1, respectively (EPA 1997b).

6.4.3.2 Benzene. (CAS No. 71-43-2; C_6H_6; Mol. wt. = 78 g/mol; WoE = A; Oral RfD = 0.003 mg/kg-day; oral SF = 0.029 mg/kg-day^{-1}; inhalation RfD = 0.00171 mg/kg-day; and inhalation SF = 0.029 mg/kg-day^{-1}). Benzene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Benzene is utilized mainly in the manufacture of ethylbenzene (intermediate in synthesis of styrene for plastics), cumene (for the manufacture of phenol and acetone), and cyclohexane (for nylon resins). Environmental emissions of benzene, which are mainly airborne, arise from gasoline vapors, auto exhaust, and industrial production and applications. The highest exposure concentrations of benzene are found in industries utilizing benzene and benzene-containing products (ADEQ 1993).

Benzene is classified as having carcinogenic and noncarcinogenic effects in humans (EPA 1997b). Toxic effects in humans from inhalation and ingestion exposures to benzene have resulted in death from respiratory arrest, CNS depression, and cardiac collapse. Inhalation exposures to humans have also resulted in hematological (deficit in the circulating blood cells, aplastic anemia, leukemia), immunological (changes in the blood levels of antibodies and circulating leukocytes), neurological (dizziness, tremor, delirium, unconsciousness), developmental (chromatid breaks, sister chromatid exchange in children of exposed females), and reproductive (impaired fertility, menstrual disorder, spontaneous abortion) effects, particularly in studies of occupationally-exposed groups. With human ingestion exposures, GI (gastritis, pyloric stenosis), hematological (decrease in erythrocytes and leukocytes), dermal (swelling and edema of skin), and neurological (vertigo, muscular incoordination, unconsciousness) effects have also been reported. Dermal exposures have resulted in skin irritation (ADEQ 1993). The inhalation NOAEL for benzene is 2.35 mg/kg-day with a critical effect of hematological impairment. The uncertainty factor for the NOAEL is 100 (EPA 1997b).

In addition, hepatic (alteration of hepatic drug metabolism), immunological (decrease in peripheral blood leukocytes), and developmental (reduction in the weight of rodent pups) effects have also been noted in animals with ingestion exposure (ADEQ 1993).

Epidemiological studies have shown an association between inhalation exposure to benzene and the development of leukemia (particularly the acute myeloid form) and lymphopoietic cancer in humans. Animal studies have supported the finding of leukemia with inhalation exposure and have also shown lymphomas with ingestion. Skin tumors have been demonstrated with dermal exposures. Genotoxic effects (chromosomal aberrations) in occupational groups have also been documented with inhalation and dermal exposures (ADEQ 1993).

6.4.3.3 Benzo(a)anthracene. (CAS No. 56-55-3; $C_{18}H_{12}$; Mol. wt = 228.3 g/mol; WoE = B2; Oral SF = 0.73 mg/kg-day^{-1}; Inhalation SF = 0.31 mg/kg-day^{-1}; Dermal SF = 0.73 mg/kg-day^{-1}). Benzo(a)anthracene is a member of a class of chemical compounds known as Polycyclic Aromatic Hydrocarbons (PAHs). PAHs are a group of chemicals formed during the incomplete burning of coal, oil and gas, garbage, and other organic substances. PAHs are used for research purposes, in medicines, and

to make dyes, plastics, and pesticides. They are found throughout the environment in air, water, and soil. PAHs can occur as a result of anthropogenic or natural activities (e.g., forest fires)(ATSDR 1990a).

PAHs tend to sorb strongly to soil and organic matter including other PAHs. Higher molecular weight PAHs tend to have lower solubilities in water. Hydrophobic PAHs have a high affinity for binding to organic matter and have relatively high biotransformation rates. The dominant mechanism of PAH removal from soil is microbial degradation. PAHs can persist in soils for years (ATSDR 1990a).

PAHs have background levels in air between 0.02 and 1.2 mg/m^3 in rural areas and between 0.15 and 19.3 mg/m^3 in urban areas. The background level of PAHs in drinking water ranges from 4 to 24 ng/L. PAHs are present in tobacco smoke, smoke from wood and creosote-treated wood products, cereals, grains, flour, bread, vegetables, fruits, meats, processed or pickled food, and beverages. The average U.S. diet contains less than 2 ppb of total PAHs (ATSDR 1990a).

Benzo(a)anthracene is a probable human carcinogen, based on sufficient data from animal bioassays in which benzo(a)anthracene produced tumors in mice exposed by gavage, intraperitoneal, subcutaneous or intramuscular injection, in addition to topical application. Although there are no human data that specifically link its exposure to human cancers, benzo(a)anthracene is a component of mixtures that have been associated with human cancer such as coal tar, soots, coke oven emissions and cigarette smoke (EPA 1997). Exposure to benzo(a)anthracene through these components may be associated with cancer of the liver, mammary gland and respiratory and gastrointestinal tracts. The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a).

6.4.3.4 Benzo(b)fluoranthene. CAS No. 205-99-2; $C_{20}H_{12}$; Mol. Wt = 252.3 g/mol; WoE = B2; Oral SF = 0.73 $mg/kg\text{-}day^{-1}$; Inhalation SF = 0.31 $mg/kg\text{-}day^{-1}$; Dermal SF = 0.73 $mg/kg\text{-}day^{-1}$). Benzo(b)fluoranthene, a member of the PAH class of chemical compounds, is a probable human carcinogen, based on sufficient data from animal bioassays in which benzo(b)fluoranthene produced tumors in mice after lung implantation, intraperitoneal or subcutaneous injection, in addition to topical application. Although there are no human data that specifically link its exposure to human cancers, benzo(b)fluoranthene is a component of mixtures that have been associated with human cancer such as coal tar, soots, coke oven emissions and cigarette smoke (EPA 1997). The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a). See Section 6.4.3.1 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.5 Benzo(g,h,i)perylene. (CAS No. 191-24-2; $C_{22}H_{12}$: Mol. Wt = 276 g/mol; WoE = D; no toxicity values available). Benzo(g,h,i)perylene, a member of the PAH class of compounds, is classified as a noncarcinogen. It is not produced commercially in the United States and has no known use (ATSDR 1990a). Benzo(g,h,i)perylene's most common route of exposure is through inhalation, but oral exposure to contaminated drinking water, food, and soil is also possible. Because of its high molecular weight, benzo(g,h,i)perylene is not as mobile as other PAHs such as phenanthrene. The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a). See Section 6.4.3.3 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.6 Chlorodifluoromethane. (CAS No. 75-45-6; $CHClF_2$; Mol. wt. = 86.47 g/mol; WoE is not available; Inhalation RfD = 14.3 mg/kg-day). Chlorodifluoromethane is a colorless, nonflammable gas with a slight etheral odor and is also known as Halocarbon 22. It is used by the semiconductor industry for plasma etching.

Chlorodifluoromethane is classified as having noncarcinogenic effects in humans from chronic exposures (EPA 1997b). Potential health effects from acute exposure to chlorodifluoromethane are through inhalation, dermal contact, and ingestion. Inhalation causes possible dizziness, drowsiness, and throat irritation at levels above 1,000 ppm. Minimal effects were observed below 1,000 ppm. Unconsciousness and death can occur at levels above 10,000 ppm. Inhalation of chlorodifluoromethane has resulted in cellular necrosis (EPA 1997b). As liquid or vapor, chlorodifluoromethane can cause eye irritation and prolonged or repeated skin contact can cause freezing of the tissue. Single dose ingestion is low to moderate, but can be aspirated into the lungs causing chemical pneumonia if vomited. The inhalation LC_{50} is 26,200 ppm/4 hours (Tech Spray 1997). The inhalation NOAEL and LOAEL are 35,370 mg/m^3 and 176,800 mg/m^3, respectively with critical effects of increased kidney, adrenal, and pituitary weights. The uncertainty factor and modifying factor for the NOAEL are 100 and 1, respectively (EPA 1997b).

6.4.3.7 Di-n-butylphthalate. (CAS No. 84-74-2; $C_{16}H_{22}O_4$; Mol. wt. = 278.35 g/mol; WoE = D; Oral RfD = 0.1 mg/kg-day). Di-n-butylphthalate is primarily used as a plasticizer for epoxy resins and polyvinyl chloride (PVC). Other applications include use as an adjusting agent for lead chromate pigments; use as a concrete additive; use in polyvinyl acetate emulsions, use as an insect repellent; and use in cosmetics (DTIC 1990a). The primary exposure pathway of concern from a soil-water systems is the migration to groundwater drinking water supplies (although this compound is strongly sorbed to soil and such migration has not been observed in the past). Inhalation resulting from volatilization from surface soils is not expected to be significant (DTIC 1990a).

Di-n-butylphthalate is classified as having noncarcinogenic effects in human from chronic exposures (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure via ingestion of di-n-butylphthalate may cause nausea, dizziness, light sensitivity, and watering and redness of the eyes. Heated vapors may irritate the eyes, nose, and throat. Accidental ingestion caused delayed effects by several hours which include: nausea, vomiting and dizziness, followed by headache, pain and eye irritation, lacrimation, photophobia and conjuctivitis. Recovery was complete within 2 weeks. Eye contact has caused immediate, severe, stinging pain, but no appreciable damage. Heated vapors may be eye, nose and throat irritant, but relatively non-irritating to the skin (DTIC 1990a).

The reproductive effects of chronic exposure to di-n-butylphthalate have not been verified; pregnancy/neonate effects include teratogen/testicular atrophy, and genotoxicity effects are negative based on limited evidence (DTIC 1990a). The oral NOAEL and LOAEL are 125 mg/kg-day and 600 mg/kg bw/day, respectively with a critical effect of increased mortality. The uncertainty factor and modifying factor for the NOAEL are 1000 mg/kg-day and 1, respectively (EPA 1997b)

6.4.3.8 Ethylbenzene. (CAS No. 100-41-4; C_8H_{10}; Mol. wt. = 106.17 g/mol; WoE = D; Oral RfD = 0.1 mg/kg-day; and inhalation RfD = 0.286 mg/kg-day). Ethylbenzene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Although ethylbenzene is used mainly as a solvent, other uses include styrene production, as well as use in asphalt, naptha, and fuels. Environmental emissions of ethylbenzene, which are mainly airborne, arise from industrial processes or in the combustion of fossil fuels. The main route of human exposure is related to chronic inhalation of low-level ethylbenzene concentrations due to direct release of ethylbenzene into the air by burning fossil fuels or using paints, inks, and insecticides containing ethylbenzene. Ingestion of ethylbenzene is also a potential route of exposure due to trace amounts found in many open water supplies.

Ethylbenzene is classified as having noncarcinogenic effects to human (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure to ethylbenzene results in neurological and respiratory depressions, in addition to eye and throat irritation. Several studies suggest that target organs

may include the liver, kidney, and hematopoietic system, although results are inconclusive (ATSDR 1990b). Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to ethylbenzene are summarized below.

Death in humans resulting from chronic, low-level exposure to ethylbenzene is unlikely (ATSDR 1990b). Ethylbenzene exhibits several systemic effects in humans. Moderate upper respiratory irritation accompanied by chest constriction has been reported in several human inhalation cases. Severe respiratory effects in humans could result following inhalation exposure to high doses of ethylbenzene. Ethylbenzene-induced hematological effects following inhalation exposure have been observed in lab animals, but these effects are unknown in humans. No hepatotoxic effects in humans have been reported. Hepatic effects in mice and rats exposed orally and by inhalation may affect humans, but studies have been inconclusive thus far. Possible renal effects (enzyme changes, organ weight increase and tubular swelling) could occur in humans exposed to high doses of ethylbenzene, but these studies are inconclusive (although animal evidence exists) (ATSDR 1990b).

Neurological effects from chronic inhalation exposure to ethylbenzene in humans is unknown. Acute inhalation at high concentrations principally affects the CNS in humans. These effects include dizziness and vertigo. Complete CNS recovery is possible following acute exposure (ATSDR 1990b).

No developmental effects has been indicated following human exposure to ethylbenzene. Animal (rat) studies indicate fetotoxic effects exist at doses which also induce toxic effects on the dams (ATSDR 1990b). No human studies are available regarding reproductive effects. Animal studies report reproductive effects, but data results were insufficient (ATSDR 1990b). Genotoxic effects of ethylbenzene in humans are not known, although two studies do suggest ethylbenzene causes an increase in the potential for genotoxicity in humans (ATSDR 1990b). The oral NOAEL and LOAEL are 97.1 mg/kg-day and 291 mg/kg-day, respectively with critical effects of liver and kidney toxicity. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b). The inhalation NOAEL and LOAEL are 434 mg/m^3 and 4340 mg/m^3, respectively with a critical effect of developmental toxicity. The uncertainty factor and modifying factor for the NOAEL are 300 and 1, respectively (EPA 1997b).

6.4.3.9 Lead. (CAS No. 7439-92-1; Pb; Mol. wt. = 207 g/mol; WoE = B2; no toxicity values available). Lead is a naturally occurring bluish-gray metal. It is used in the production of batteries, ammunition, various metal products (such as sheet lead, solder, brass and bronze products, and pipes) and in ceramic glazes and paints. Tetraethyl lead and tetramethyl lead were historically used as a gasoline additive, but these uses were discontinued in 1996 (ATSDR 1997).

Lead is classified as a probable human carcinogen based on sufficient evidence in animals, but inadequate evidence in humans (EPA 1997b). Acute exposure to lead can occur primarily through ingestion, inhalation and dermal contact. Exposure may cause CNS depression, weakness in extremities, and increased blood pressure. Exposure to elevated concentrations may cause liver and neurological toxicity (ATSDR 1997).

Lead produces neurotoxic and behavioral effects particularly in children. However, the EPA believes that it is inappropriate to set an RfD for lead and its inorganic compounds because the agency believes that some of the effects may occur at such low concentrations as to suggest no threshold. The EPA has also determined that lead is a probable human carcinogen (classified at B2) (ASTM 1995).

6.4.3.10 Mercury. (CAS No. 7439-97-6; Hg; Mol. wt. = 201 g/mol; WoE = D; Oral RfD = 0.0003 mg/kg-day, inhalation RfD = 0.0000857 mg/kg-day). The chemistry of mercury in the environment is complex, not only because of its various oxidation states but also because of biotic and

abiotic methylation and demethylation processes, complexation with organic and inorganic ligands, and the differential solubility and volatility of various forms. Speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of mercury compounds.

Although the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (Wren 1986; Scheuhammer 1987).

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates, e.g., methyl and metal radicals, which cause cellular damage (Wren 1986; Scheuhammer 1987; Manzo et al. 1992). The inhalation NOAEL and LOAEL are none and 0.009 mg/m^3, respectively with critical effects of hand tremor, increases in memory disturbances, and slight subjective and objective evidence of autonomic dysfunction. The uncertainty factor and modifying factor for the NOAEL are 30 and 1, respectively (EPA 1997b).

6.4.3.11 Phenanthrene. (CAS No. 85-01-8; $C_{14}H_{10}$; Mol. wt. = 178.08 g/mol; WoE =D; no toxicity values available). Phenanthrene, a member of the PAH class of chemical compounds, is noncarcinogenic, producing negative cancer results when tested. Phenanthrene is virtually insoluble in water, but is degraded by microbes in the soil. Phenanthrene is not produced commercially in the United States and there is no known use for phenanthrene except as a research chemical. The oral NOAEL is 150 mg/kg-day with critical effects of the gastrointestinal, hepatic, and renal systems. The NOAEL was done for a rat for four days (ATSDR 1990a). See Section 6.4.3.1 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.12 Phenol. (CAS No. 108-95-2; C_6H_6O; Mol. wt. = 94.11 g/mol; WoE = D; Oral RfD = 0.6 mg/kg-day). Phenol is primarily used as a chemical intermediate in the synthesis of organic chemicals (primarily phenolic resins). Less significant uses of phenol involve use as a solvent in petroleum refining and as a disinfectant (DTIC 1990a). The primary pathway of concern from soil-water systems is the migration of phenol to groundwater supplies of drinking water. Data suggests that such migration has occurred in the past. The consumption of fish or other organisms is not expected to be a significant route of exposure (DTIC 1990a).

Phenol is classified as having noncarcinogenic effects to humans from chronic exposures and does not exhibit carcinogenic effects in humans (EPA 1997b). Chronic effects include liver and kidney damage, in addition to skin discoloration. Pregnancy/neonate effects are teratogenic only at maternally lethal doses; studies show fetotoxic effects occur at doses that are not toxic to the female animal during pregnancy, and genotoxic results are unclear due to conflicting evidence (DTIC 1990a). Phenol is readily absorbed from all routes of entry, distributed throughout the body, metabolized and rapidly excreted. The most frequent adverse effects from phenol reported in humans resulted from skin contact. Signs and symptoms can develop rapidly with serious consequences including shock, convulsions, cyanosis, coma and death. Direct contact with the skin results in chemical burns (DTIC 1990a).

Acute exposure to phenol has a marked corrosive action on tissue. On contact with the eyes, it may cause severe damage and blindness. On skin, it induces anesthesia and blanching of the exposed area. If not removed promptly, it may cause a severe burn and systemic intoxication. Systemic effects, which can result from any route of exposure, include paleness, weakness, sweating, headache, ringing of the ears, cyanosis, shock, excitement, frothing of the nose and mouth, and death (DTIC 1990a). The oral NOAEL

and LOAEL are 60 mg/kg-day and 120 mg/kg-day, respectively with a critical effect of reduced fetal body weight in rats.

6.4.3.13 Tetrachloroethene. (CAS No. 127-18-4; C_2Cl_4; Mol. wt. = 165.85 g/mol; WoE = C-B2; Oral RfD = 0.01 mg/kg-day; oral SF = 0.052 mg/kg-day^{-1}; inhalation SF = 0.00203 mg/kg-day^{-1}). Tetrachloroethene (PCE) is used primarily in the dry cleaning industry. It is also used in cold cleaning, vapor degreasing of metals, and as a chemical intermediate in the synthesis of fluorocarbons. Minor applications include various manufacturing and industrial processes as well as medicinal uses (DTIC 1990a).

The primary exposure pathway of concern from a soil-water system is the migration of PCE to groundwater used as sources for drinking water. Inhalation resulting from volatilization from surface soils and drinking water may also be important (DTIC 1990a).

PCE is classified as a possible human carcinogen. Chronic effects include liver and kidney toxicity, and both pregnancy/neonate effects and genotoxicity effects are negative (DTIC 1990a). Acute exposure via ingestion and inhalation can cause nausea, vomiting, headache, dizziness, drowsiness, and tremors. Skin contact with liquid causes irritation and blistering. Both the liquid and vapor are eye irritants (DTIC 1990a). The oral NOAEL and LOAEL are 14 mg/kg-day and 100 mg/kg-day, respectively with critical effects of hepatotoxicity in mice and weight gains in rats. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b).

6.4.3.14 Toluene. (CAS No. 108-88-3; $C_6H_5CH_3$; Mol. wt. = 92.15 g/mol; WoE = D; Oral RfD = 0.2 mg/kg-day; and inhalation RfD = 0.114 mg/kg-day). Toluene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Toluene is used as a solvent in paint and paint thinners, in addition to various printing and leather tanning processes. Environmental air emissions of toluene arise from automobile exhaust, in addition to petroleum, iron-coke, and styrene production. Toluene can enter soil, surface water, and ground water from spills and leaking underground storage tanks. The main route of human exposure is related to inhalation of low-level toluene concentrations due to the direct release of toluene into the air, soil, surface water, and ground water.

Toluene is classified as having noncarcinogenic effects in humans (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute inhalation exposure to toluene results primarily in respiratory tract irritation. High concentrations of toluene under chronic exposure in rats indicated respiratory irritation and pulmonary lesions (ATSDR 1994). Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to toluene are summarized below.

Acute inhalation exposure at elevated concentrations resulted in heart rhythm alterations when tested on animals. Chronic exposure studies in humans indicated possible gastrointestinal irritation. Hematological effects are not expected to occur in humans due to the lack of response in several animal studies. Hepatic effects have not been observed in humans, but there is a possibility that the liver's ability to metabolize xenobiotics similar to toluene is affected. Renal effects have not been observed in workers exposed to low level, chronic exposure to toluene (Askergren *et al.*, 1981; Neilsen *et al.*, 1985). Eye and skin irritations have been reported after human exposure to toluene (ATSDR 1994).

Animal studies indicate immunological effects such as decreased thymus weight, lymphocyte culture responses and antibody plaque-forming cell responses, but no human data is currently available (ATSDR 1994). The primary neurological effect of inhalation exposure in humans is CNS depression with the following symptoms: fatigue, confusion, and incoordination, as well as impairment in reaction time, perception, and motor control. High concentration, occupational exposure has led to residual or

permanent CNS effects. Toluene is ototoxic in both animals and humans. Hearing deficits were identified when workers were chronically exposed to 100 ppm of toluene (ATSDR 1994). It is difficult to speculate whether reproductive effects occur in women exposed to toluene (ATSDR 1994).

Human developmental effects are inconclusive, but the occurrence of neurobehavioral effects and possible fetotoxicity in animals is a cause of concern to humans (ATSDR 1994). Genetic assays generally indicate that toluene is nonmutagenic and nongenotoxic (ATSDR 1994). The oral NOAEL and LOAEL are 223 mg/kg-day and 446 mg/kg-day, respectively with critical effects of changes in liver and kidney weights. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b). The inhalation NOAEL and LOAEL are none and 332 mg/m^3, respectively with a critical effect of neurological effects. The uncertainty factor and modifying factor for the NOAEL are 300 and 1, respectively (EPA 1997b).

6.4.3.15 TPH-Diesel. (CAS No. none; TPH is a mixture; Mol. wt. = none; WoE not available; Oral RfD = 0.6 mg/kg-day). Diesel is a blue liquid mixture used mainly as a fuel in machinery and automobiles. Diesel is incompatible with oxygen and strong oxidizing agents, and will ignite in the presence of heat, sparks, or flame. Acute effects from overexposure are mild eye irritation, severe skin irritation, respiratory and gastrointestinal irritation. When diesel burns, the exhaust when inhaled causes short term carboxyhemoglobinemia and long term lung cancer in humans (Phillips 66 1997). The oral LD_{50} in rats is 9 mL/kg and dermal LD_{50} in rabbits is >5 mL/g (Phillips 66 1997).

Total Petroleum Hydrocarbons as diesel (TPH-diesel) is comprised of hydrocarbons in the C_{10} to C_{20} range. Because of their higher molecular weights, constituents in these products are less volatile, less water soluble, and less mobile than gasoline-range hydrocarbons. About 25 to 35% of No. 2 fuel oil is composed of aromatic hydrocarbons, primarily alkylated benzenes and naphthalenes. The BTEX concentrations are generally low (ASTM 1995).

6.4.3.16 1,1,1-Trichloroethane. (CAS No. 71-55-6; $C_2H_3Cl_3$; Mol. Wt = 133.42 g/mol; WoE not available; Oral RfD = 0.02 mg/kg-day; Inhalation RfD = 0.286 mg/kg-day). 1,1,1-Trichloroethane (1,1,1-TCA) is widely used as a cleaning solvent because of its nonflammability and solvency properties. As of 1985, approximately 28% of the total production was used in vapor degreasing and 41% in cold cleaning. Common solvent uses include cleaning of electrical equipment, motors, electronic components and instruments, missile hardware, photographic film, printed circuit boards, upholstery, and various metal and plastic parts during manufacture. It is also used as a solvent for adhesives and coatings, photoresist polymers, textile dyes, as coolant and lubricant in metal cutting oils ,as a component in inks and drain cleaners, and as a chemical intermediate in the production of vinylidene chloride. It has minor use in aerosols where it acts both as a vapor pressure depressant and as a solvent and carrier for the active ingredients (DTIC 1990b).

1,1,1-TCA is expected to be fairly mobile in the soil/groundwater system, particularly is soils of low organic carbon where adsorption is low. Volatilization is an important removal process for near-surface contamination (DTIC 1990b).

1,1,1-TCA is classified as having noncarcinogenic effects in humans from chronic exposure (EPA 1997b) and does not exhibit carcinogenic effects in humans. Chronic effects include liver toxicity. Pregnancy/neonate effects are negative and genotoxic effects are unclear due to conflicting results (DTIC 1990b). Acute exposure leads to dizziness, drowsiness, lack of coordination, increased reaction time and irregular heart beat. Both liquid and vapor are eye irritants. Skin contact may produce dermatitis. Depression of the central nervous system is the primary toxic effect in humans who have been subjected to short-term, high-level inhalation exposure. Inhalation of 450 ppm for 8 hr caused eye, nose, and throat irritation. Acute inhalation exposures can also adversely effect the cardiovascular system.

Numerous deaths have been attributed to deliberate or occupational inhalation to 1,1,1-TCA. In the majority of human fatalities, death results from CNS depression, edema and pulmonary congestion (DTIC 1990b). The single oral dose NOAEL is 1400 mg/kg-day with a critical effect of depressed hepatic metabolism. The uncertainty factor for the NOAEL is 100 (EPA 1997b).

6.4.3.17 Xylenes, mixed. (CAS No. 1330-20-7; C_8H_{10}; Mol. wt. = 106 g/mol; WoE = D; Oral RfD = 2 mg/kg-day). Xylene is an aromatic hydrocarbon which consists of three isomers (ortho, meta, and para). Xylene occurs naturally in the environment, in addition to a product of human activity. Xylene is a product of petroleum, coal, and forest fires. Xylene is used as a solvent in the printing, rubber, and leather industries. Xylene is also found in airplane fuel, gasoline, and cleaning agents. Xylene is used as a material in the paint, chemical, plastic, and synthetic fiber industries, in addition to an ingredient in the coating of fabrics and papers. Xylene emissions occur primarily from industrial sources, auto exhaust, and solvent use. Release from the use, storage, and transport of petroleum and xylene products also serves as possible routes for human exposure (ATSDR 1990c).

Xylene is classified as having noncarcinogenic effects in humans (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure to high concentrations of xylene can cause irritation to the skin, eyes, nose, and throat; breathing difficulty; impaired pulmonary function; delayed response to visual stimulus; impaired memory; stomach discomfort; and possible hepatic and nephrotic effects. Both acute and chronic exposure to high concentrations of xylene can cause CNS effects such as dizziness, headaches and general confusion. Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to xylene are summarized below (ATSDR 1990c).

Animal studies show observed hepatic, nephrotic, pulmonary, cardio, and CNS effects. Chronic, low-level concentrations of Xylene have not been studied in depth. Carcinogenic effects on animals have not been determined, although xylene is thought to be a possible human teratogen. Primary target organs: liver and CNS (ATSDR 1990c).

Xylene can be fatal to both humans and animals following inhalation and oral exposure. No fatal dermal exposure cases have been reported in humans. Death in humans and animals appears to be caused by either respiratory failure or ventricular fibrillation. The amount of xylene necessary to cause death is relatively large in both animals and humans, and reports of death in humans following inhalation occurred in areas of poor ventilation. Therefore, it is highly unlikely that inhalation or ingestion of the small amounts of xylene present in contaminated water or air would pose a fatal risk (ATSDR 1990c).

In humans, acute inhalation of xylene produced nose and throat irritation. Severe lung congestion with pulmonary hemorrhages and edema was noted in a worker who died following acute inhalation. In addition, chronic occupational exposure to xylene vapors was associated with labored breathing and impaired pulmonary function. Symptoms such as nausea, vomiting and gastric discomfort have been noted in workers following inhalation (ATSDR 1990c).

Available human studies suggest possible evidence that inhalation exposure to solvent mixtures containing xylene may increase the risk of developing renal dysfunction or renal damage resulting in increased blood urea concentrations, decreased urinary clearance of endogenous creatinine, increased lysozymuria, increased urinary levels of B-glucuronidase, and increased urinary excretion of albumin, erythrocytes, and leukocytes. No human data is available regarding the renal toxicity of xylene following oral or dermal exposure. Human dermal exposure causes irritation, dryness, scaling, and vasodilation of the skin.

Results of experimental studies with animals provide further evidence that mixed xylene and individual isomers are neurotoxicants following inhalation exposure. Neurotoxicity symptoms in animals include narcosis, prostration, incoordination, tremors, muscular spasms, labored breathing, behavioral changes, hyperactivity, elevated auditory thresholds, hearing loss, changes in brain enzyme activity and changes in brain protein levels (ATSDR 1990c).

Various assays indicate that mixed xylene and xylene isomers are nongenotoxic. No data were available regarding the development of cancer in humans following inhalation, oral, or dermal exposure to mixed xylene or xylene isomers (ATSDR 1990c). The oral NOAEL is 179 mg/kg-day with critical effects of hyperactivity, decreased body weight, and increased mortality (males). The uncertainty factor and modifying factor for the NOAEL are 100 and 1, respectively (EPA 1997b).

6.4.3.18 Radionuclides. The EPA classifies all radionuclides as "Group A" carcinogens (i.e., WoE =A) because radionuclides emit ionizing radiation and because of the extensive weight-of-evidence provided by epidemiological studies of radiation-induced cancers in humans. Ionizing radiation has sufficient energy to interact with matter and produce an ejected electron and a positively charged ion. In addition, ionizing radiation can produce new chemical species, known as free radicals, from water in the body. Free radicals are highly reactive and may combine with other elements or compounds within a cell to produce toxins or otherwise disrupt a cell's chemical balance. These disruptions may result in mutations or other deleterious effects.

Radionuclides are characterized by the type and energy level of the radiation emitted. Radionuclides contained in WAG 4 soils produce external radiation exposures principally through the production of beta, gamma, and alpha radiation.

The general health effects of radiation can be divided into stochastic and nonstochastic effects (i.e., those health effects not related to threshold dose and those related to threshold dose). Developing cancer from exposure to any amount of radiation is a stochastic effect. Examples of nonstochastic effects include acute radiation syndrome and cataract formation, both of which occur only at high levels of exposures.

Radiation can damage cells in different ways. First, radiation can cause damage to the strands of genetic material, DNA, in a cell. The cell may not be able to recover from this type of damage, or the cell may live on in a functionally abnormal condition. If the abnormally functioning cell divides and reproduces, a tumor or mutation in the tissue may develop. The rapidly dividing cells that line the intestines and the stomach and the cells that make blood in the bone marrow are very sensitive to this kind of damage. Organ damage results from the damage caused to the individual cells. This type of damage has been reported with doses of 10 to 500 rem. Acute radiation sickness is seen only after doses of greater than 50 rem. This dose is usually only received by personnel in proximity to serious nuclear accidents. Principal adverse effects associated with exposure to ionizing radiation are carcinogenicity, mutagenicity, and teratogenicity.

When cells damaged by radiation are reproductive cells, genetic damage can occur in the offspring of the person exposed. The developing fetus is especially sensitive to radiation. The type of malformation that may occur is related to the stage of fetal development and the cells that are differentiating at the time of exposure. Radiation damage to children exposed while in the womb is related to the dose that the pregnant mother received. Mental retardation is another possible effect of fetal radiation exposure.

In the following subsections f_1 is the fractional absorption of a stable radionuclide from the gastrointestinal tract and the class D, W, and Y describes the clearance time of inhaled radioactive

materials from the lung. The class D applies to a half-time of less than 10 days, class W applies to a half-time from 10 to 100 days, and class Y applies to a half-time of greater than 100 days. The following subsections provide additional information about the specific radionuclide COPCs at WAG 4.

6.4.3.18.1 Americium-241. (CAS No. 014596-10-2; Atomic No. 95; Mol. wt. = 241 g/mol; Half-life = 432 years; Ingestion SF = 3.28E-10 risk/pCi; inhalation SF = 3.85E-08 risk/pCi; and external SF = 4.59E-09 risk/pCi). Am-241 is produced by the beta decay of Pu-241. This isotope has been distributed widely in the environment as a result of nuclear weapons fallout. Am-241 decays by alpha emission, which makes the isotope important for internal exposure, whether it is ingested or inhaled. The alpha decay is accompanied by emission of gamma of radiation 60 keV with an abundance of 36%, which is of concern where Am-241 is concentrated, but is not important at environmental levels. The International Committee on Radiological Protection (ICRP) has assigned a value of 5.00E–04 to f_1, the fractional absorption of americium from the gastrointestinal tract, for all compounds of americium. For inhalation exposures, the ICRP recommends assigning all compounds of americium to inhalation Group W. Most (90%) of the americium entering the blood stream is deposited in the liver and the bone, with only a small amount being deposited in human reproductive organs. The biological half-lives in the liver and the bone are 40 and 100 years, respectively. The amount deposited in reproductive organs is considered to remain permanently.

6.4.3.18.2 Actinium-228. (CAS No. 014331-83-0; Atomic No. 89; Mol. wt. = 228 g/mol; Half-life = 6.13 hours; Ingestion SF = 1.62E-12 risk/pCi; inhalation SF = 3.27E-11 risk/pCi; and external SF = 3.28E-06 risk/pCi). Ac-228 is a beta-emitting member of the decay chain of naturally occurring Th-232. It has a half-life of 6.13 hours and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain.

ICRP (1979) has assigned a value of 0.001 to f_1, the fractional absorption of actinium from the gastrointestinal tract. Oxides and hydroxides are assigned by ICRP to inhalation class Y, halides and nitrates are assigned class W, and other common compounds are assigned class D. Actinium primarily deposits in the mineral bone and the liver, with biological half-lives of 100 years and 40 years, respectively.

6.4.3.18.3 Barium-133. (CAS No. 013981-41-4; Atomic No. 56; Mol. wt. = 133 g/mol; Half-life = 10.5 years; Ingestion SF = 2.70E-12 risk/pCi; inhalation SF = 4.03E-12 risk/pCi; and external SF = 9.15E-07 risk/pCi). Ba-133 has a physical half-life of 10.53 years and decays by electron capture to cesium-133. ICRP has assigned a value of 0.1 to f_1, the fractional absorption of barium from the gastrointestinal tract. All common compounds are assigned inhalation class D by ICRP. Barium primarily deposits in the bone, and ICRP assumes that it is distributed throughout the volume of mineral bone.

6.4.3.18.4 Bismuth-212. (CAS No. 014913-49-6; Atomic No. 83; Mol. wt. = 212 g/mol; Half-life = 60.55 minutes; Ingestion SF = 6.20E-13 risk/pCi; inhalation SF = 3.65E-11 risk/pCi; and external SF = 6.67E-07 risk/pCi). Bi-212 is an alpha-and beta-emitting member of the decay chain associated with Th-228, which is part of the larger decay chain of naturally occurring Th-232. Bismuth-212 has a physical half-life of 60.5 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.05 to f_1, the fractional absorption of bismuth from the gastrointestinal tract. Bismuth nitrate is assigned by ICRP to inhalation class D, and all other compounds are assigned class W. Bismuth is primarily deposited in the kidneys, with different fractions assumed to clear with biological half-lives of 0.6 and 5 days.

6.4.3.18.5 Bismuth-214. (CAS No. 014733-03-3; Atomic No. 83; Mol. wt. = 214 g/mol; Half-life = 19.9 minutes; Ingestion SF = 1.95E-13 risk/pCi; inhalation SF = 1.46E-11 risk/pCi; and external SF = 6.02E-06 risk/pCi). Bi-214 is an alpha- and beta-emitting member of the decay chain associated with Ra-226, which is part of the larger decay chain of naturally occurring U-238. Bismuth-214 has a physical half-life of 19.7 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.05 to f_1, the fractional absorption of bismuth from the gastrointestinal tract. Bismuth nitrate is assigned by ICRP to inhalation class D, and all other compounds are assigned class W. Bismuth is primarily deposited in the kidneys, with different fractions assumed to clear with biological half-lives of 0.6 and 5 days.

6.4.3.18.6 Cesium-137. (CAS No. 010045-97-3; Atomic No. 55; Mol. wt. = 137 g/mol; Half-life = 30.2 years; Ingestion SF = 3.16E-11 risk/pCi; inhalation SF = 1.91E-11 risk/pCi; and external SF = 2.09E-06 risk/yr-pCi/g). Cs-137 is a fission product produced in nuclear reactors and in nuclear weapons detonations. Cs-137 is rapidly absorbed into the bloodstream and distributes throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and energetic beta and gamma radiation from its daughter, Ba-137m, result in essentially whole-body irradiation (Amdur et al. 1991). The radioactive half-life of Cs-137 is 30 years. Its biological half-life in adults is 50 to 150 days, in children, 44 days. Cs-137 exists in secular equilibrium with Ba-137m, which is the major contributor to the dose received from a 0.662 MeV gamma ray. The critical organ for Cs-137 exposure is the whole body.

6.4.3.18.7 Europium-152. (CAS No. 014683-23-9; Atomic No. 63; Mol. Wt = 152 g/mol; Half-life = 13.6 years; Ingestion SF = 5.73E-12 risk/pCi; inhalation SF = 7.91E-11 risk/pCi; and External SF = 4.08E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.4.3.18.8 Lead-212. (CAS No. 015092-94-1; Atomic No. 82; Mol. wt. = 212 g/mol; Half-life = 10.6 hours; Ingestion SF = 1.80E-11 risk/pCi; inhalation SF = 3.85E-11 risk/pCi; and external SF = 3.00E-07 risk/pCi). Pb-212 is a beta-emitting member of the decay chain associated with Th-228, which is of part of the larger decay chain of naturally occurring Th-232. Pb-212 has a physical half-life of 10.64 hours and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.2 to f_1, the fractional absorption of lead from the gastrointestinal tract. All commonly occurring compounds of lead are assigned by ICRP to inhalation class D. Lead is primarily deposited in the bone, and to a lesser extent in the liver and kidneys, with different fractions assumed to clear with biological half-lives of 12, 180, and 10,000 days.

6.4.3.18.9 Plutonium-238. (CAS No. 013981-16-3; Atomic No. 94; Mol. wt. = 238 g/mol; Half-life = 87.8 years; Ingestion SF = 2.95E-10 risk/pCi; inhalation SF = 2.74E-08 risk/pCi; and external SF = 1.94E-11 risk/pCi). Pu-238 is produced in reactors and is used in space power systems. It has been released to the environment due to burnup during atmospheric re-entry. Pu-238 has a radioactive half-life of 87.7 years, and decays by alpha emission. ICRP has assigned a value of 1E-05 to f_1, the fractional absorption of plutonium from the gastrointestinal tract, for oxides and hydroxides. Oxides and hydroxides are assigned by ICRP to inhalation class Y. All other commonly occurring compounds of plutonium are assigned by ICRP to inhalation class W, with a value of 1E-04 for f_1, the fractional absorption of plutonium from the gastrointestinal tract.

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones (ATSDR 1989). Biological half-lives in liver and bone are assumed by ICRP as 40 years and 100 years, respectively. For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

6.4.3.18.10 Plutonium-239 (for Pu-239/240). (CAS No. 015117-48-3; Atomic No. 94; Mol. wt. = 239 g/mol; Half-life = 24,100 years; Ingestion SF = 3.15E-10 risk/pCi; inhalation SF = 2.78E-08 risk/pCi; and external SF = 1.87E-11 risk/pCi). The main source of plutonium in the environment is from nuclear-weapons testing, with smaller contributions from accidents and space power systems burnup in the atmosphere. U.S. soil contains an estimated 5E-02 pCi/g of plutonium in the top 5 cm (4 in.).

Pu-239 has a radioactive half-life of 2.41E+04 years. Pu-239 decays by alpha emission, thus its mode of decay is accompanied by emission of x- and gamma radiation that are low energy and do not contribute significantly to radiation dose at environmental levels. ICRP has assigned a value of 1E-05 to f_1, the fractional absorption of plutonium from the gastrointestinal tract, for oxides and hydroxides. Oxides and hydroxides are assigned by ICRP to inhalation class Y. All other commonly occurring compounds of plutonium are assigned by ICRP to inhalation class W, with a value of 1E-04 for f_1, the fractional absorption of plutonium from the gastrointestinal tract.

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones (ATSDR 1989). Biological half-lives in liver and bone are assumed by ICRP as 40 years and 100 years, respectively. For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

6.4.3.18.11 Radium-226. (CAS No. 013982-63-3; Atomic No. 88; Mol. wt. = 226 g/mol; Half-life = 1,600 years; Ingestion SF = 2.95E-10 risk/pCi; inhalation SF = 2.72E-09 risk/pCi; and external SF = 1.31E-08 risk/pCi). Ra-226 is an alpha-emitting member of decay chain of naturally occurring U-238. Ra-226 has a physical half-life of 1622 years. ICRP has assigned a value of 0.2 to f_1, the fractional absorption of radium from the gastrointestinal tract. All radium compounds are assigned by ICRP to inhalation class W. Radium is primarily deposited in the bone, and is assumed by ICRP to be distributed throughout the volume of mineral bone.

6.4.3.18.12 Silver-108m D. (CAS No. 014391-65-2m D; Atomic No. 47; Mol. Wt = 108 g/mol; Half-life = 127 years; Ingestion SF = 6.05E-12 risk/pCi; inhalation SF = 7.02E-11 risk/pCi; and External SF = 5.62E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.4.3.18.13 Thallium-208. (CAS No. 014913-50-9; Atomic No. 81; Mol. wt. = 208 g/mol; Half-life = 3.05 minutes; Ingestion SF = 1.75E-14 risk/pCi; inhalation SF = 1.36E-14 risk/pCi; and external SF =1.45E-05 risk/pCi). Tl-208 is a beta-emitting member of the decay chain associated with Th-228, which is of part of the larger decay chain of naturally occurring Th-232. Tl-208 has a physical half-life of 3.1 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 1.0 to f_1, the fractional absorption of thallium from the gastrointestinal tract. Oxides, hydroxides, halides, and nitrates are assigned by ICRP to inhalation class W, and all other commonly occurring compounds are assigned class D. ICRP assumes bismuth is deposited throughout all organs and tissues of the body, with a biological half-life of 10 days.

6.4.3.18.14 Uranium-234, -235, -238. (CAS No. 013966-29-5, 007440-61-1, respectively; Atomic No. 92; Mol. wt. = 234, 235, and 238 g/mol, respectively; Half-lives = 245,000 years, 704,000,000 years, and 4,470,000,000 years, respectively; Ingestion SF = 4.44E-11 risk/pCi, 4.27E-11 risk/pCi, respectively; inhalation SF =1.4E-8 risk/pCi, 1.3E-8 risk/pCi, and 1.24EE-8 risk/pCi, respectively; and external SF = 2.14E-11 risk/yr/pCi/g, 2.63E-7 risk/yr/pCi/g, and 1.5E-11 risk/yr/pCi/g, respectively). Natural uranium contains three isotopes: U-234, U-235, and U-238. The percent abundance of each isotope in natural uranium is, respectively, 0.006% and 99.27% (ATSDR 1990d). Uranium can be found in the earth's crust at an average concentration of 2 ppm. The ambient air concentration of uranium in the United States ranges from 0.3 to 0.011 fCi/m^3 (1fCi = 10^{-3} pCi). The

concentration in drinking water ranges from 0.07 to 653 pCi/L with a median value of 0.1 to 0.2 pCi/L. The average daily intake of uranium has been established to be 0.007 pCi/day from air (0.01 mg/day), 0.7 to 1 pCi/day from food (1 to 1.4 mg/day), and 0.6 to 2.0 pCi/day (0.83 to 2.78 mg/day) from drinking water.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity, and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha radiation that is unable to penetrate skin, but can travel short distances in the body if uranium is inhaled or ingested. Natural uranium emits very small amounts of gamma radiation that can penetrate the skin; therefore, little, if any, danger exists from this type of radiation from uranium (ATSDR 1990d). Moreover, no human or animal studies have definitively linked inhalation of or oral exposure to natural uranium to the development of cancer.

For noncancer health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air, and soil does not appear to have any toxic effects. Animals that have had oral ingestion of, inhalation of, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected.

The only significant systemic health risk in humans from exposure to nonenriched uranium is potential damage to the kidneys. However, epidemiological studies have not noted an increase in deaths from urogenital or renal diseases, and intravenous studies have failed to identify significant damage to human kidneys following exposure to uranium (ATSDR 1990d). Overall, studies in animals and humans also indicate that exposure to uranium is unlikely to produce immunological or neurological effects. Although the data are conflicting, animal studies indicate that exposure to uranium may affect fetal weight and skeletal development in animals, and may possibly alter the ratio of male to female live births in areas where people have excessive exposure to uranium (ATSDR 1990d). With the exception of soluble salts, no oral or inhalation RfDs are available for uranium on IRIS or HEAST, nor has ATSDR established minimum risk levels for different environmental media (EPA 1995b; ATSDR 1990d).

ICRP has assigned a value of 0.05 to f_1, the fractional absorption of uranium from the gastrointestinal tract, for soluble fluorides and nitrates. The assigned inhalation class for fluorides and nitrates is class D. Less soluble fluorides and oxides are assigned a value of 0.05 to f_1, and an inhalation class W. Highly insoluble oxides are assigned a value of 0.002 to f_1, and an inhalation class Y.

Uranium is deposited primarily in the kidney and bones. Biological half-lives in kidney for different deposition fractions are assumed by ICRP as 6 days and 1500 days, respectively. Biological half-lives in bone for different deposition fractions are assumed by ICRP as 20 days and 5000 days, respectively. ICRP assumes that uranium-234 and 238 are uniformly distributed throughout the volume of mineral bone.

6.4.3.18.15 Zirconium-95. (CAS No. 013967-71-0; Atomic No. 40; Mol. Wt = 95 g/mol; Half-life = 0.175 years; Ingestion SF = 3.92E-12 risk/pCi; inhalation SF = 6.48E-12 risk/pCi; and External SF = 2.81E-06 risk/yr-pCi/g). See Section 6.4.3.19 for a general discussion of the chemical properties and toxicity of radionuclides.

6.5 Risk Characterization

Risk characterization involves estimating the magnitude of the potential adverse effects, summarizing the nature of the potential threats to public health, and evaluating the weight-of-evidence supporting risk estimates and the magnitude of uncertainty associated with those estimates. Specifically,

risk characterization involves combining the results of the exposure and toxicity assessments to provide quantitative estimations of risk. The risk characterization methods described in this Section are based on standard EPA guidance (EPA 1989a). An analysis of the uncertainties associated with the risk estimates calculated in this BRA is provided in Section 6.6, Uncertainty Analysis.

To characterize potential carcinogenic risks, probabilities that an individual will develop cancer over a lifetime of exposure to Site COPCs are estimated from projected intakes and chemical-specific dose-response information. To characterize potential noncarcinogenic risks, comparisons are made between estimates of intakes of Site COPCs and toxicity values. These methodologies, and results of the risk characterization for the WAG 4 retained sites and COPCs, are discussed in the sections below.

6.5.1 Generalized Approach

To quantify human health risks, contaminant intakes are calculated for each COPC by way of each applicable exposure route (see Section 6.3 and Tables D-24 through D-35). As discussed in Section 6.3, these contaminant intakes are based on measured concentration estimates at each retained release site. To determine human health risks, the contaminant specific intakes are compared to the applicable chemical-specific toxicity data discussed in Section 6.4. The following subsections discuss the equations that are used to calculate risks for each retained site.

6.5.1.1 Carcinogenic Health Effects. Cancer risks related to the Site are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the Site carcinogenic COPCs. The risk of cancer from exposure to carcinogens is estimated by using the cancer slope factor to covert chemical intake averaged over a lifetime of exposure directly to an incremental risk of an individual developing cancer. Because the cancer slope factor is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data, the carcinogenic risk estimate will generally be an upper-bound estimate. This means that there is reasonable confidence that the "true risk" will not exceed the cancer risk estimated and is likely to be less than predicted.

The cancer risks calculated represent excess cancer risks that may be experienced in a lifetime under a given exposure scenario. The term "excess" refers to levels above the background cancer risk. For example, national cancer statistics indicate that each person has approximately a one-in-three chance, or 333,333 chances in one million, of developing cancer during his lifetime (ACS 1986). An individual with an excess cancer risk of one in a million (denoted as either 1E-06 or 1 x 10^{-6}) has a total cancer risk of 333,334 in one million of developing cancer: 333,333 chances per million from background exposures, plus one chance per million from exposure to the Site.

The following calculations are used to obtain numerical estimates, (i.e., unitless probability) of lifetime cancer risks:

$$Risk = Intake \times SF \qquad (6\text{-}18)$$

where

Risk = potential lifetime cancer risk (unitless)

SF = slope factor, for chemicals (mg/kg/day)-1, or radionuclides (pCi)-1

Intake = chemical intake (mg/kg/day), or radionuclide intake (pCi).

To develop a total risk estimate for a given release site, the contaminant risks are summed for each COPC at the site.

$$Risk_T = \Sigma Risk_i \quad (6\text{-}19)$$

where

$Risk_T$ = total cancer risk, expressed as a unitless probability

$Risk_i$ = risk estimate for the *i*th contaminant.

Similarly, the risk values for each exposure route are summed to obtain the total cancer risk for each potential carcinogen.

Permissible excess cancer risks cover a range of values. The National Contingency Plan adopted an excess cancer risk range of 1E-06 to 1E-04 (i.e., one in one million to one in ten thousand) as an acceptable risk range. Consistent with this range is recent EPA guidance which states that remediation is generally not required for excess cancer risks less than 1E-04 (EPA 1991c). In addition, several past regulatory decisions indicate that in many circumstances, excess cancer risks greater than 1E-04 are permissible (Travis and Hattemer-Frey 1988).

6.5.1.2 Noncarcinogenic Effects. Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating hazard quotients. The potential for health effects associated with exposure to noncarcinogens is evaluated by comparing an exposure level over a specified time period (e.g., lifetime) with a reference dose derived for a similar exposure period. This ratio of toxicity is called a hazard quotient (HQ). The HQ is the ratio of the intake rate to the RfD, as follows:

$$HQ = Intake/RfD \quad (6\text{-}20)$$

where

HQ = noncancer hazard quotient (unitless)

Intake = chemical intake (mg/kg/day)

RfD = reference dose (mg/kg/day).

The hazard index (HI) is used to determine if potential noncancer effects may be of concern; it does not predict the incidence or severity of potential health effects. HIs are calculated by summing the HQs for each chemical across all exposure routes. The HI is calculated using the following equation:

$$HI = \Sigma \frac{Intake_i}{RfD_i} \quad (6\text{-}21)$$

where

HI = hazard index (unitless)

$Intake_i$= exposure level (intake) for the *i*th toxicant (mg/kg/day)

RfD_i = reference dose for the *i*th toxicant (mg/kg/day).

In the above equation, intake and RfD are expressed in the same units and represent the same exposure time period.

A HI greater than 1.0 indicates that there may be concern for potential noncancer health effects; however, it does necessarily mean that health effects will occur. As a general rule, the greater the HI is above 1.0, the greater the level of concern.

It is important to note that the level of concern does not increase linearly as the threshold level of 1.0 is approached or exceeded because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects (i.e., the slopes of the dose-response curves in excess of the RfD can range widely depending on the substance). For this BRA, to provide conservative estimates of HIs, individual HQs will be assumed to be additive, regardless of toxic effects or mechanisms of action.

6.5.2 Estimates of Human Health Risk

Estimates of WAG 4 human health risks during each evaluated time period (see Section 6.3 for a discussion of exposure time periods) are presented in Tables D-43 through D-48 (Appendix D) and Figures 6-5 through 6-9. For each time period, carcinogenic risks and noncarcinogenic HIs are shown in separate tables and figures.

As discussed in Section 6.3, risk and HI estimates for the air and groundwater pathway exposure routes (i.e., inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, dermal absorption of groundwater, inhalation of water vapor from indoor water use) are calculated in a cumulative manner in accordance with *Guidance Protocol for the Performance of Cumulative Risk Assessments at the INEL* (LMITCO 1995). Potential risks are estimated for COPCs in air and groundwater on a WAG-wide, instead of site-specific, basis. As a result, the estimated risk for each COPC in air and groundwater is assumed to be the same for each site retained for evaluation in the BRA. For example, the potential risk from inhalation of Cs-137 at CFA-04 is the same as the potential risk from inhalation of Cs-137 at CFA-07. Likewise, the potential future residential risk from ingestion of 1,1,1-trichloroethane at CFA-04 is the same as the potential future residential risk from ingestion of this COPC at CFA-07. This method of assessing risks for air and groundwater exposure routes is required because releases via these routes are generally not isolated and may affect all sites within the WAG. The WAG 4 air and groundwater pathway cumulative risk results are presented below in Section 6.5.2.1.

Conversely, soil exposure routes (i.e., incidental soil ingestion, dermal contact with soil, ingestion of homegrown produce) are not assessed in a cumulative manner because chemical exposures via these routes are generally isolated to a specific site (i.e., exposures are site-specific) and do not affect exposures at other sites. Site-specific risk results for WAG 4 are presented below in Section 6.5.2.2.

Risk and HQ estimates for ingestion of groundwater containing maximum predicted COPC concentrations are shown in Tables D-49 and D-50. These risk estimates are presented separately because maximum predicted COPC concentrations may occur beyond the exposure time periods evaluated in the BRA.

The following sections summarize the site-specific excess cancer and noncancer risk estimates for the current occupational worker, future occupational worker, and future resident. As discussed in

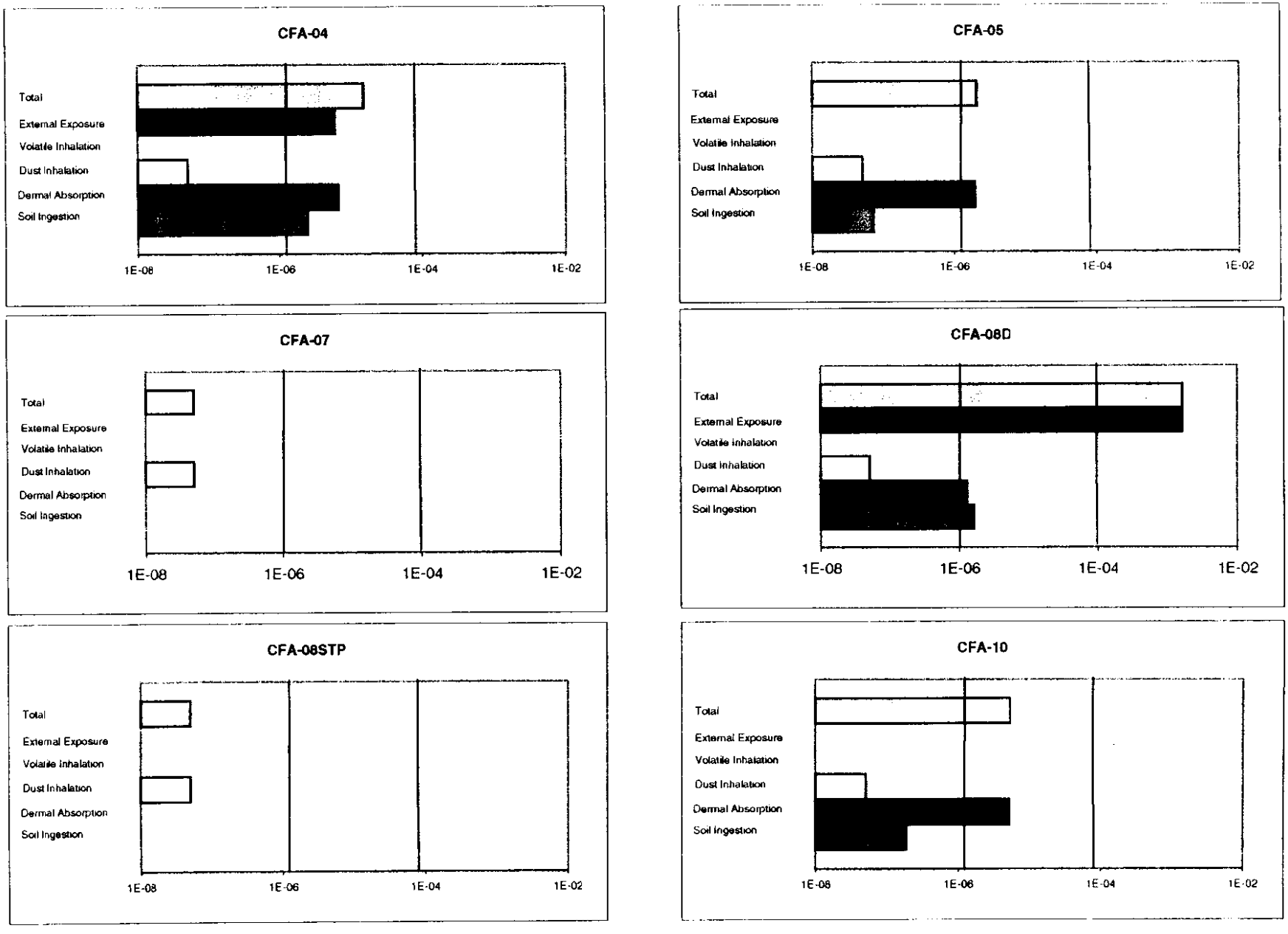


Figure 6-5. Total risk for worker at 0 years.

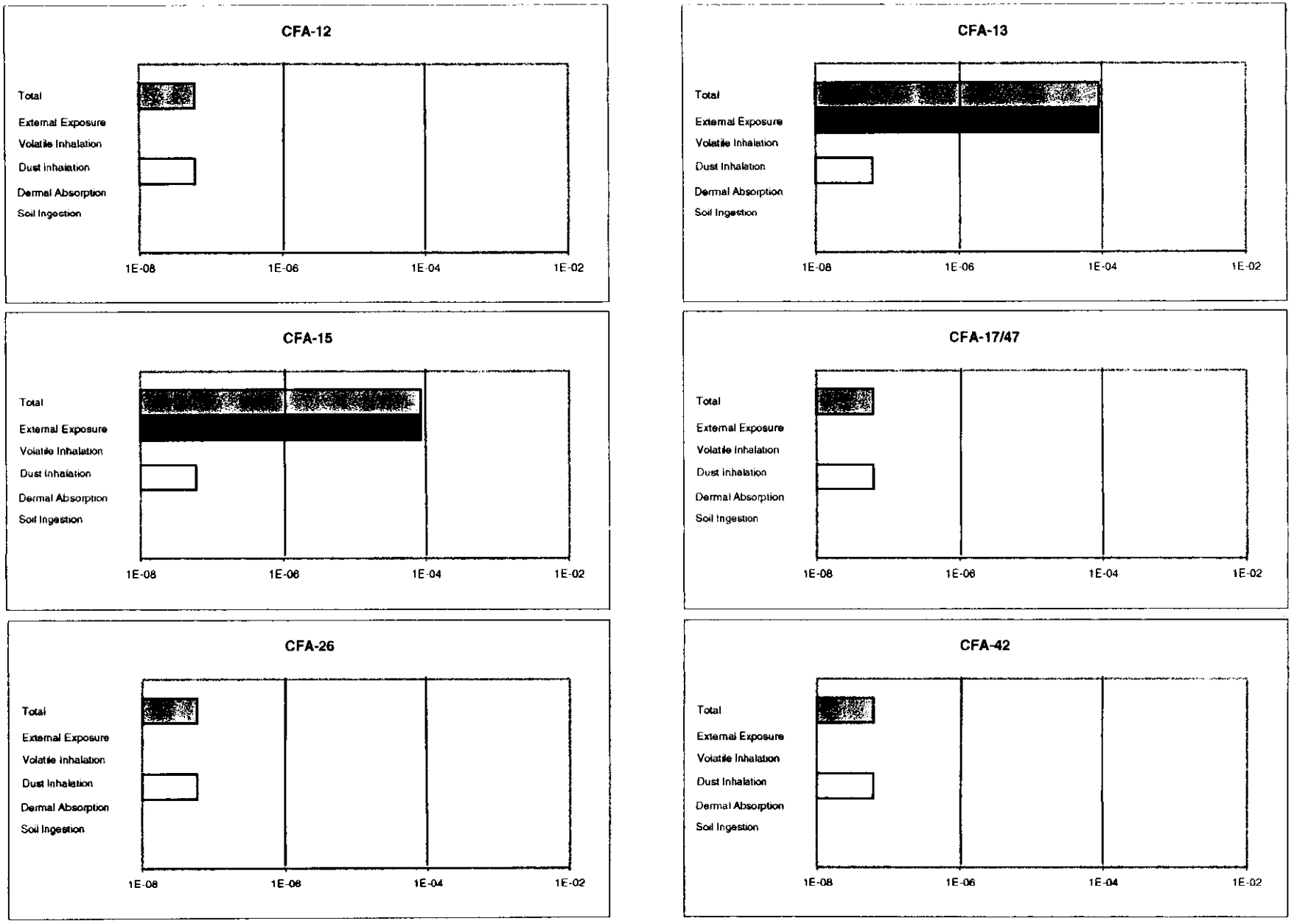
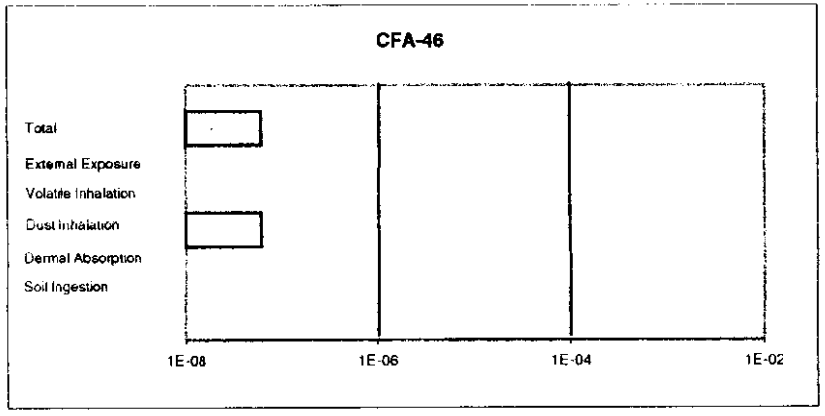
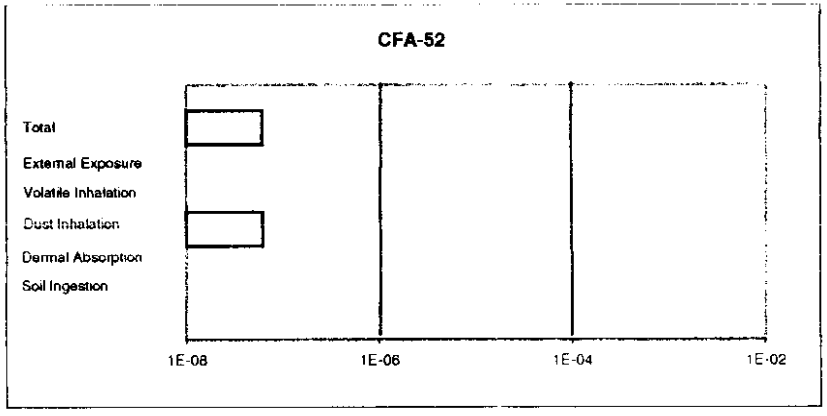


Figure 6-5. (continued).





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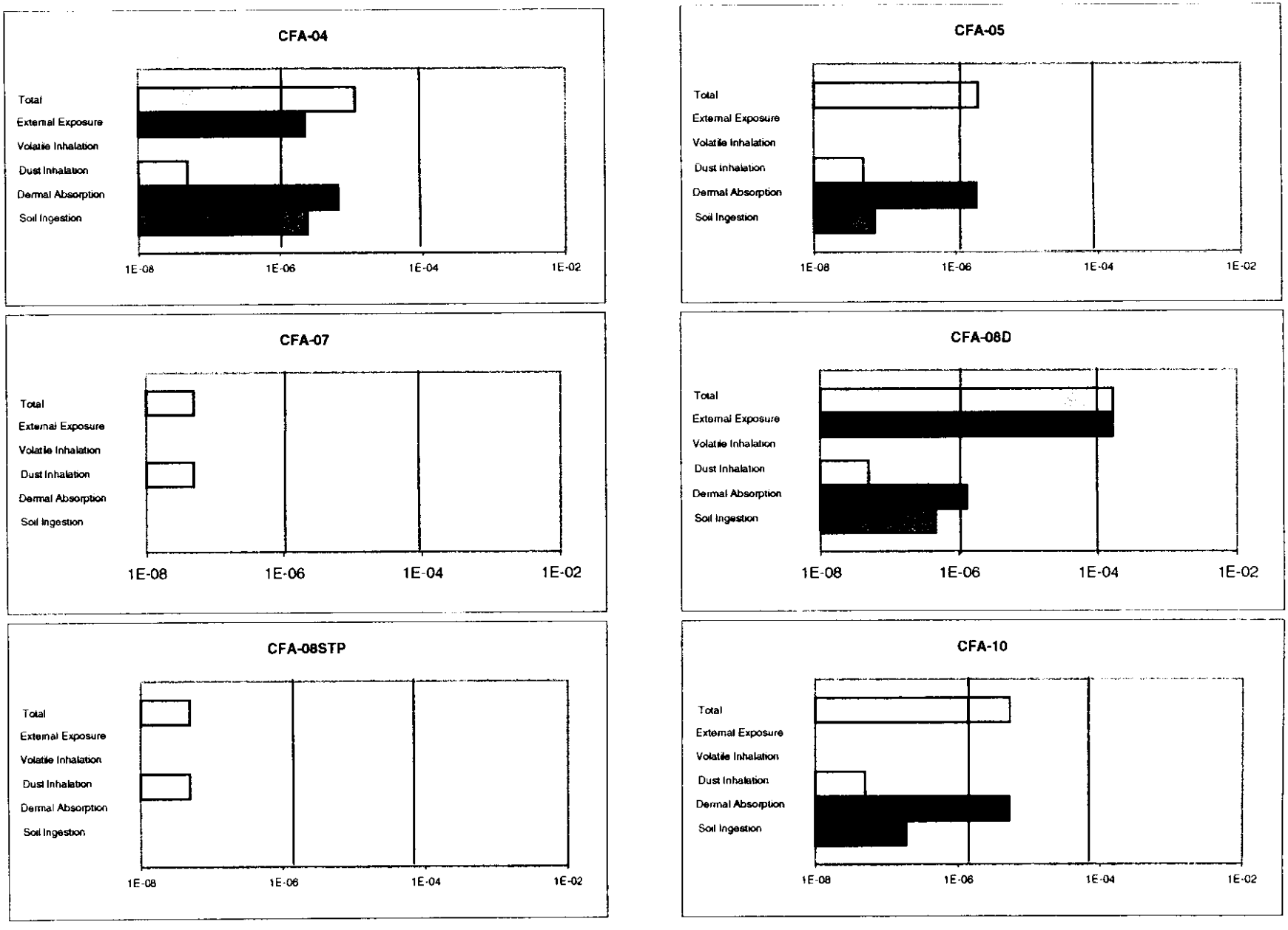


Figure 6-6. Total risk for worker at 100 years.

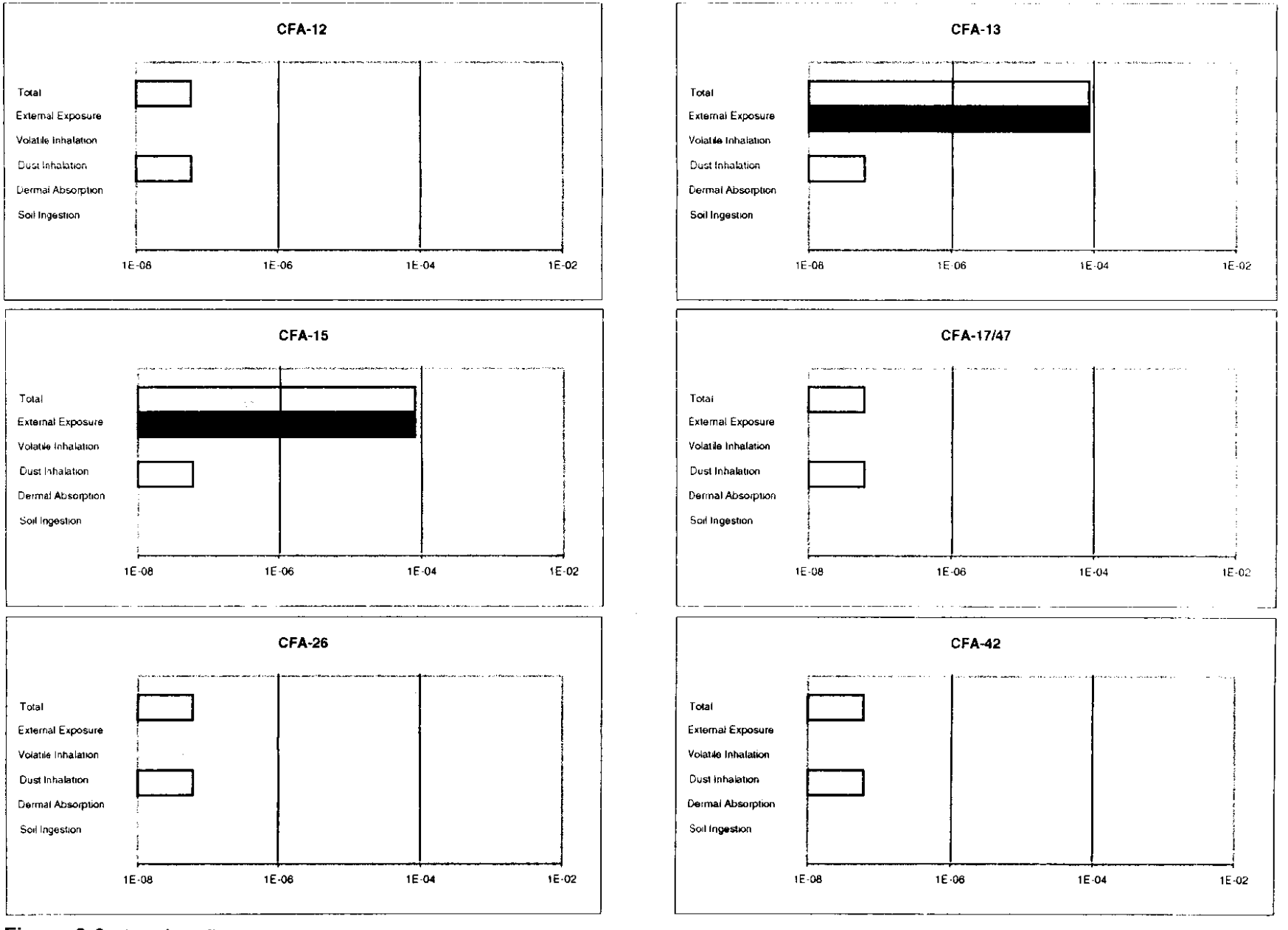
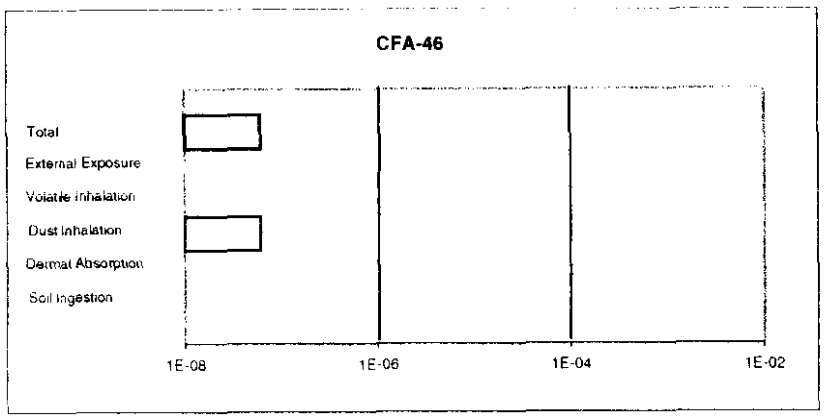


Figure 6-6. (continued).



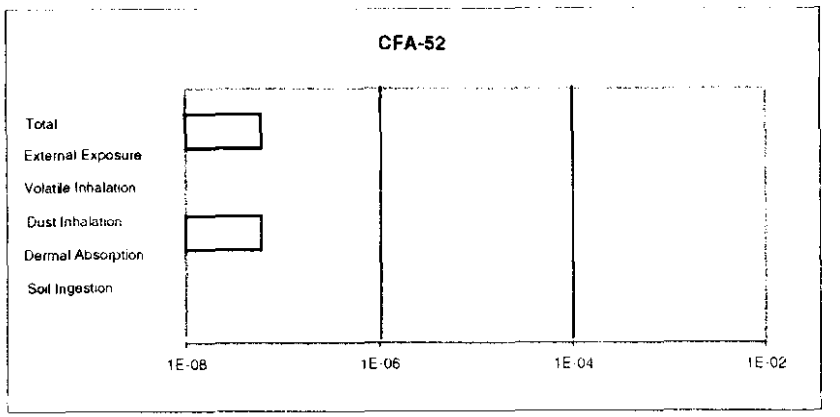


Figure 6-6. (continued).

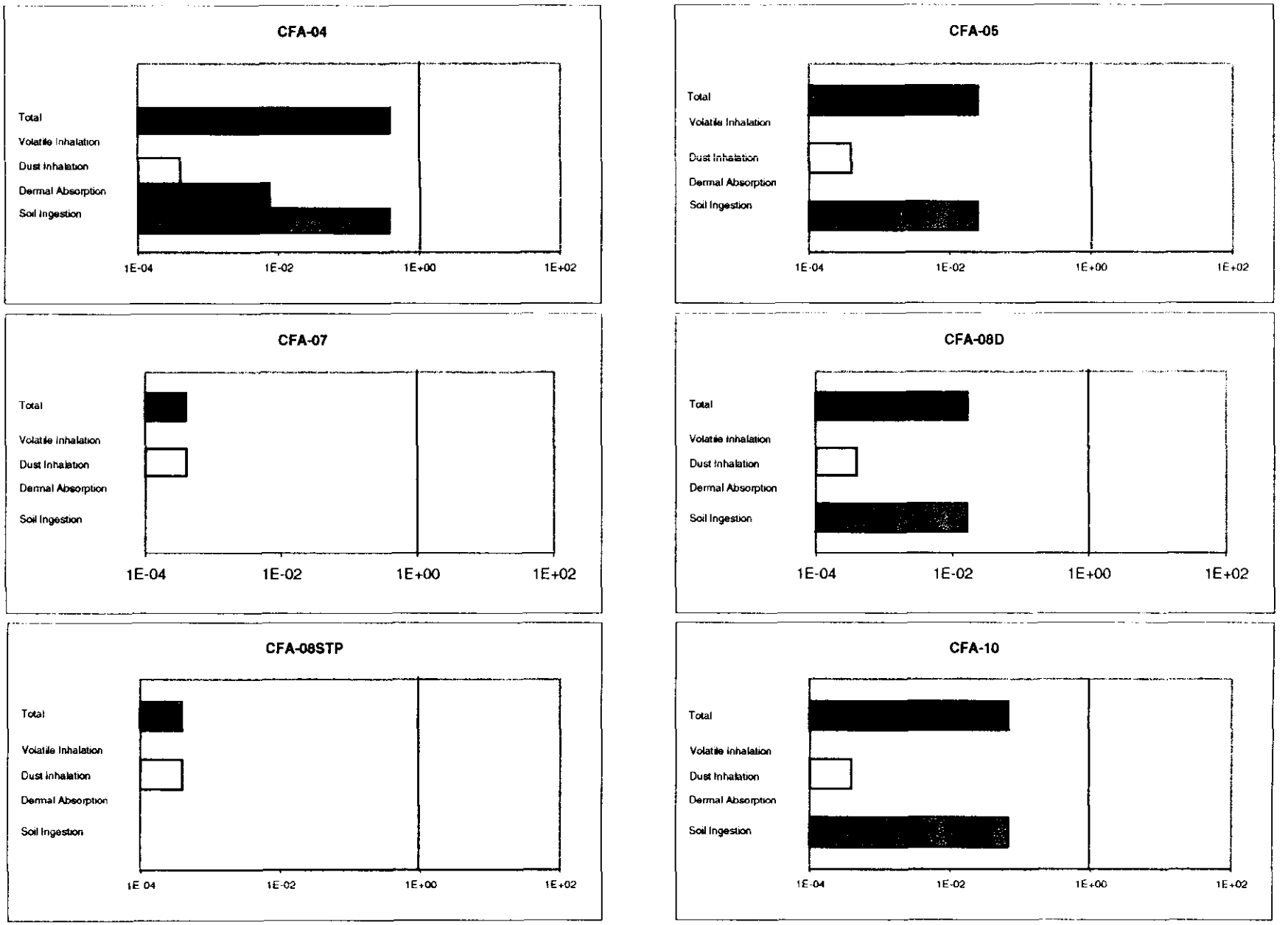
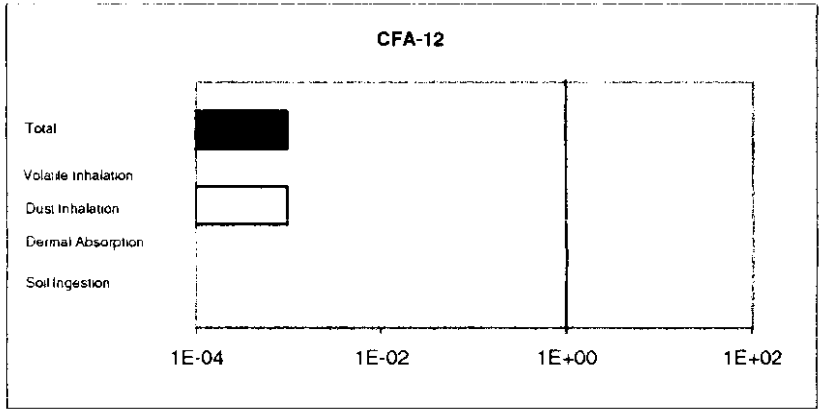
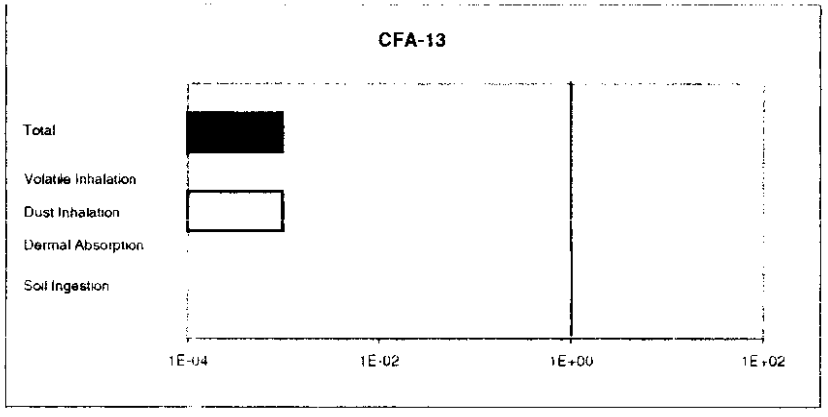
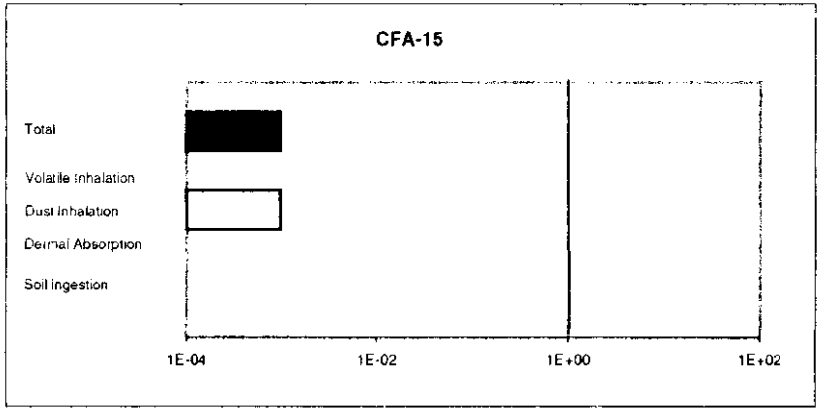
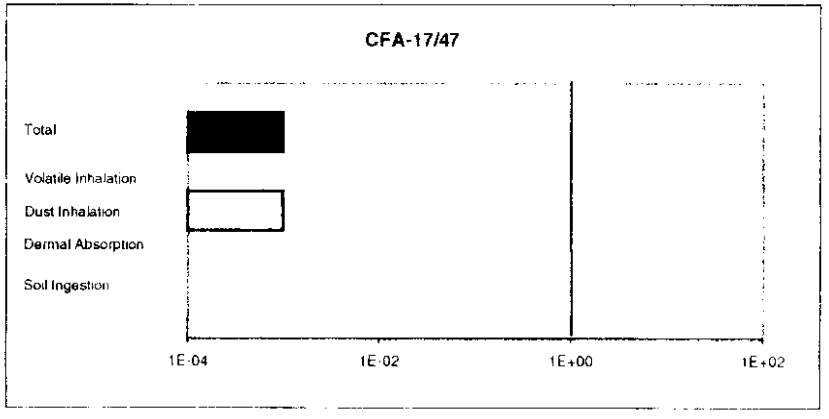


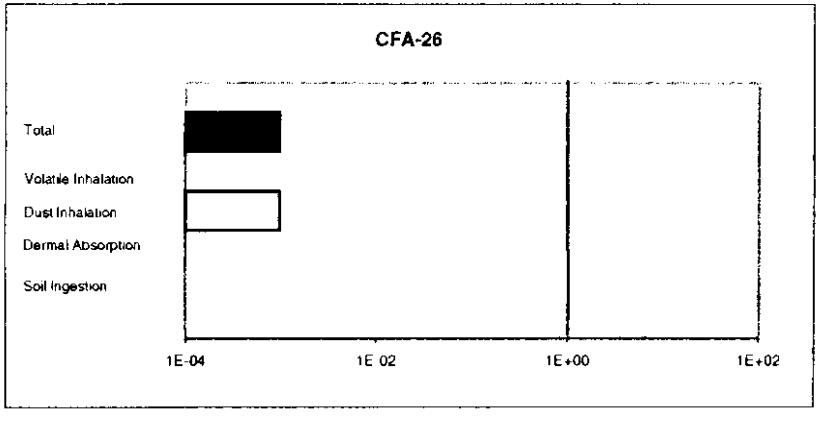
Figure 6-7. Total hazard indices for worker at 0 years.











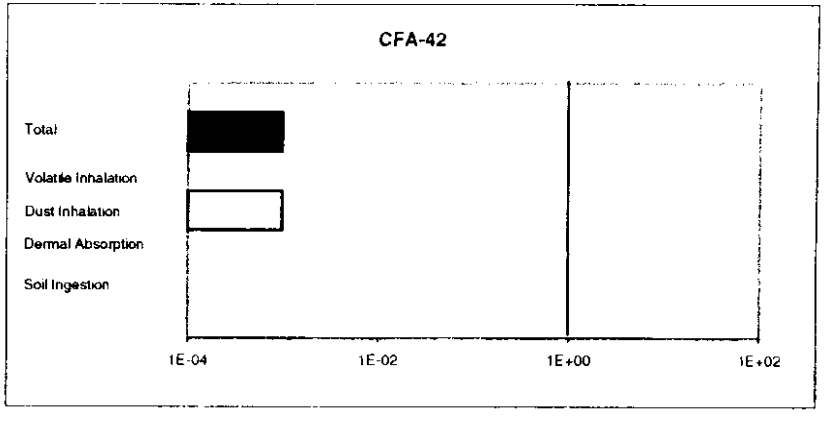
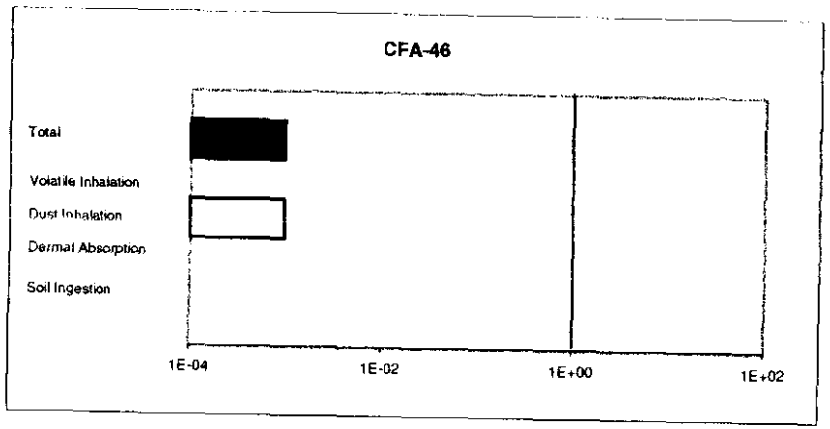


Figure 6-7. (continued).



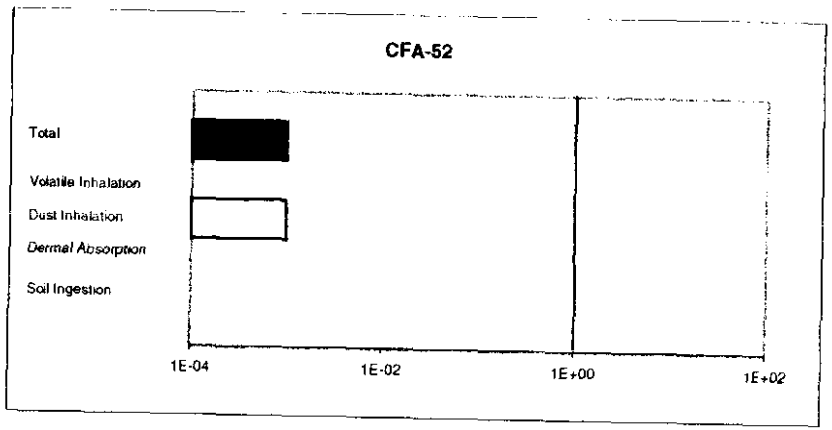


Figure 6-7. (continued).

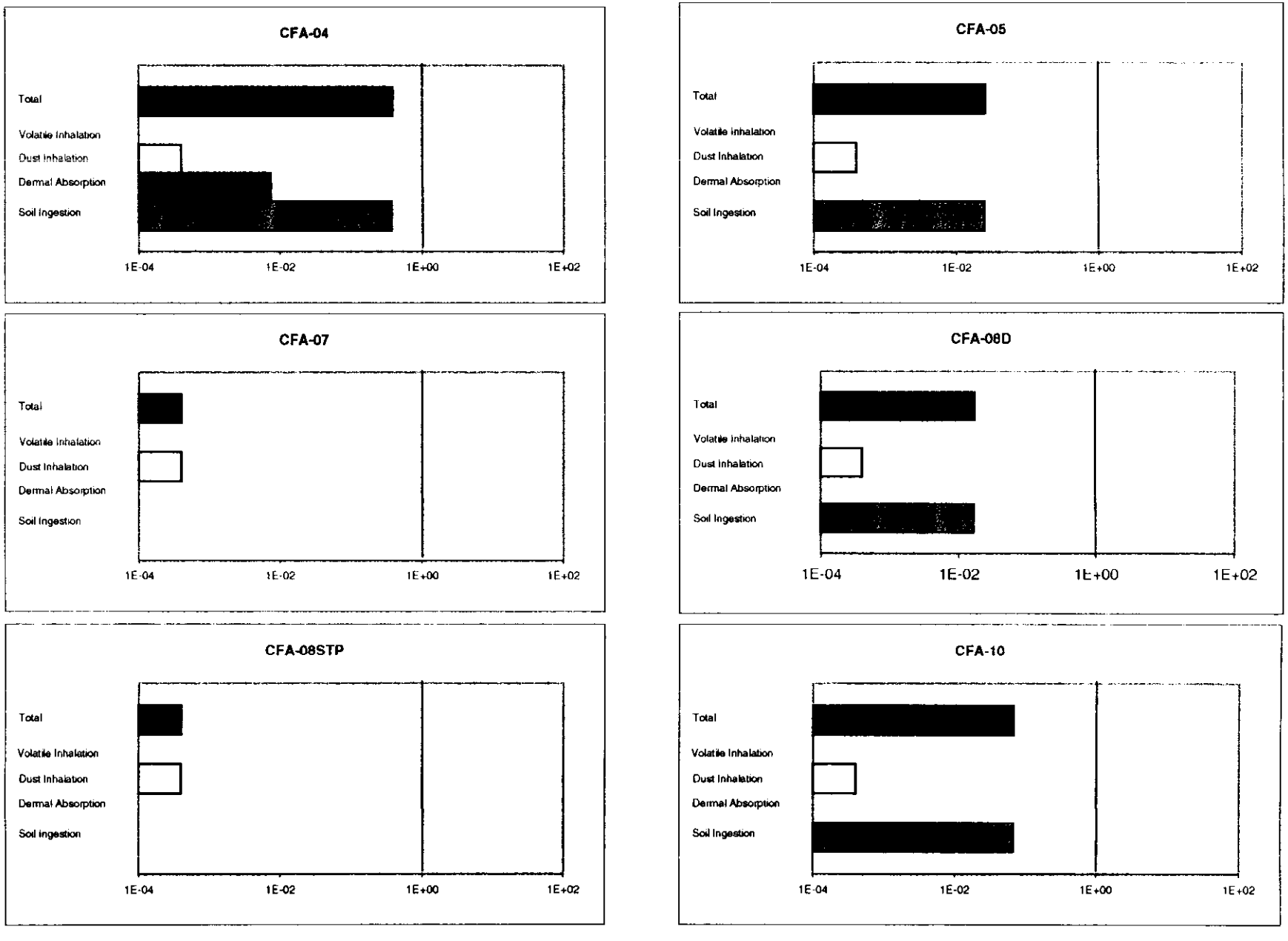


Figure 6-8. Total hazard indices for worker at 100 years.

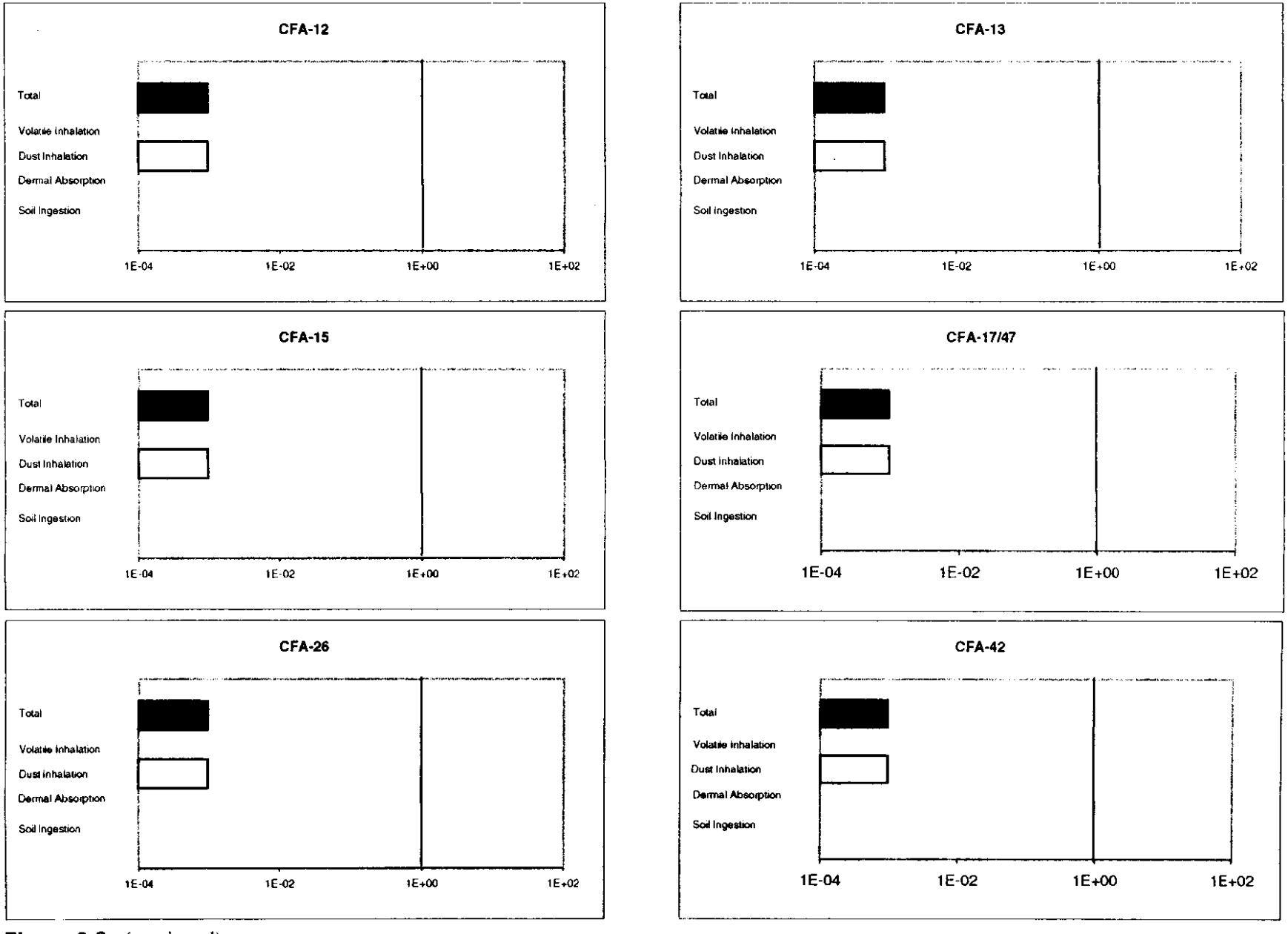
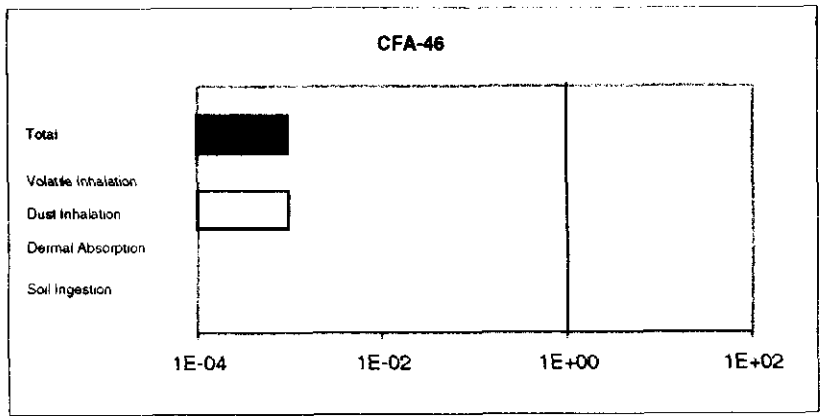


Figure 6-8. (continued).



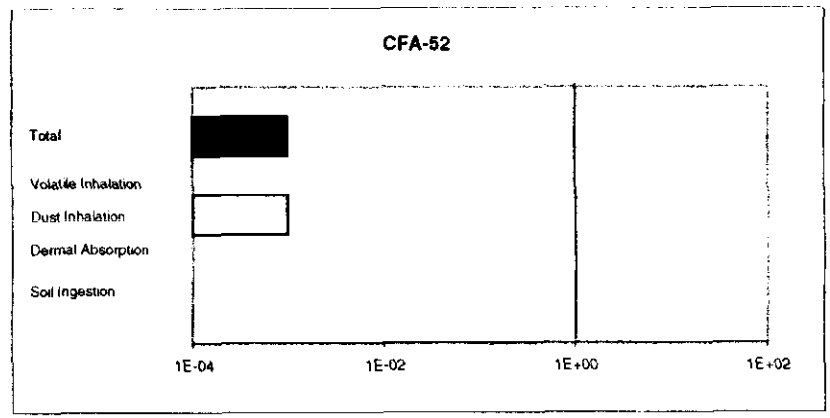


Figure 6-8. (continued).

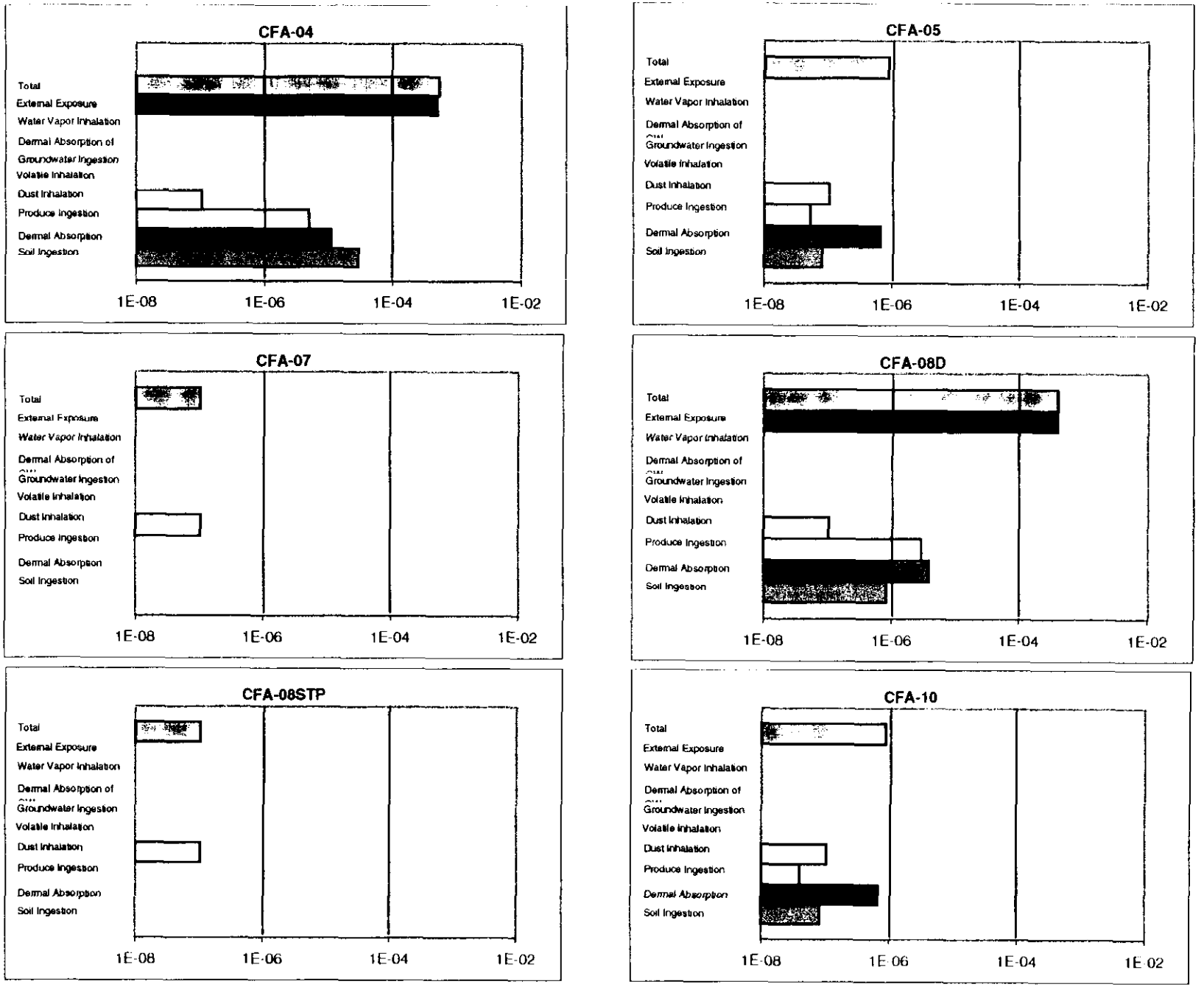
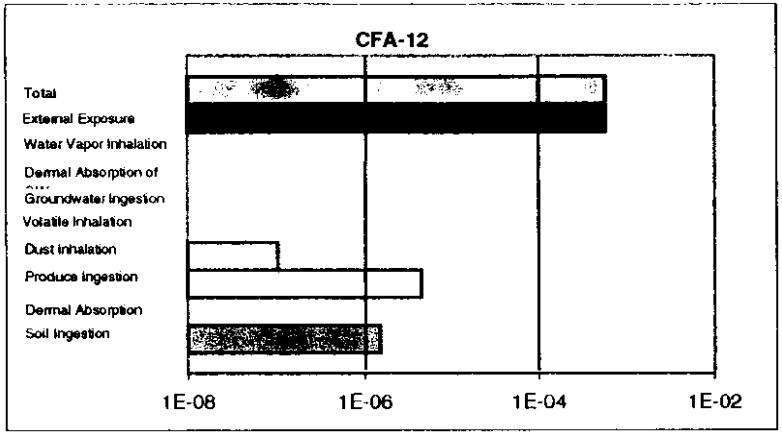
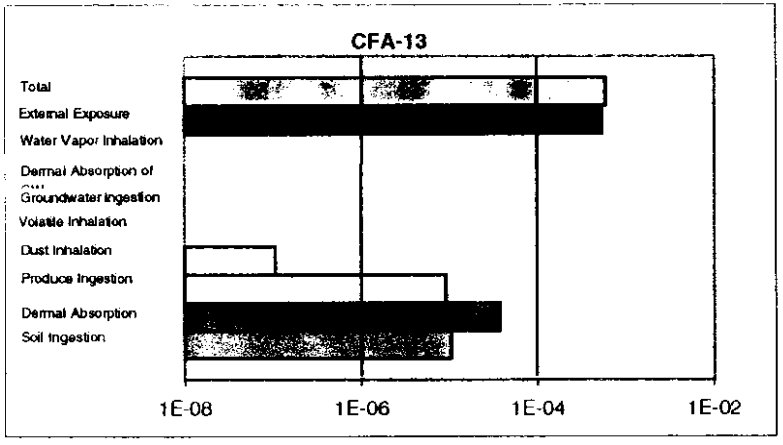
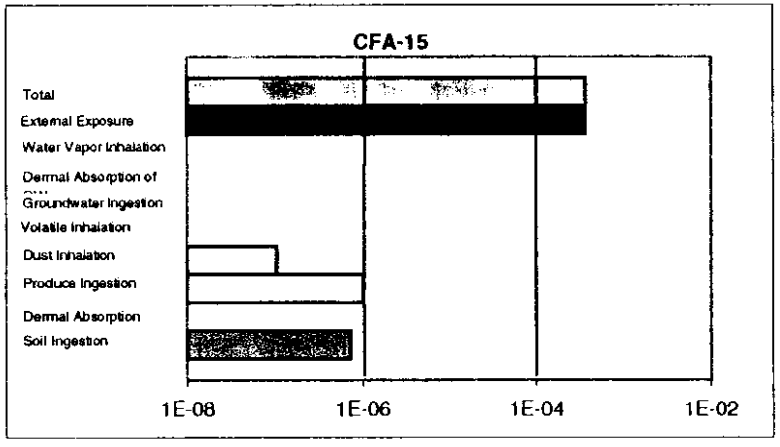
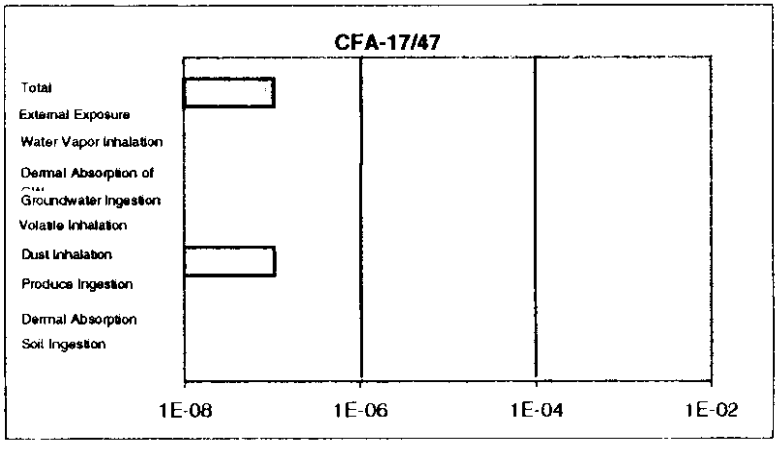


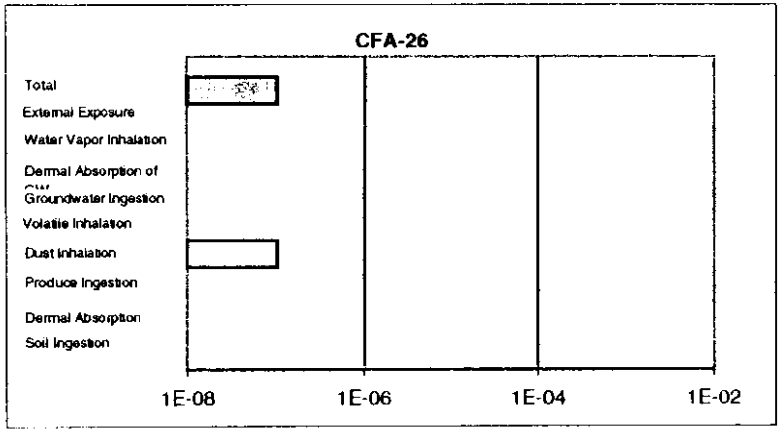
Figure 6-9. Total risk for resident at 100 years.











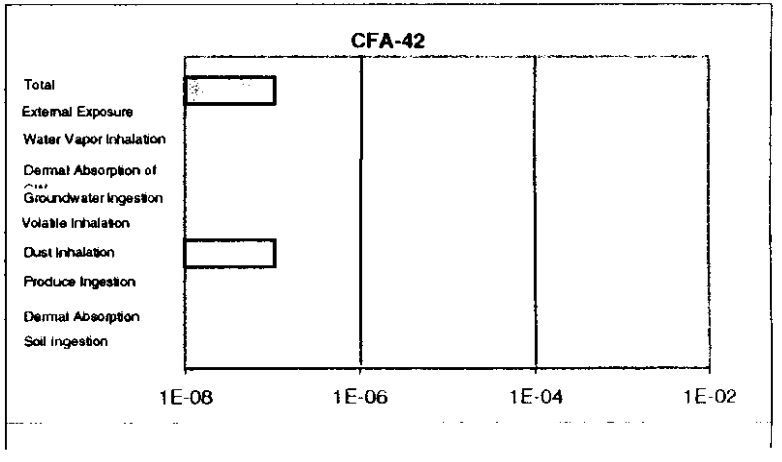
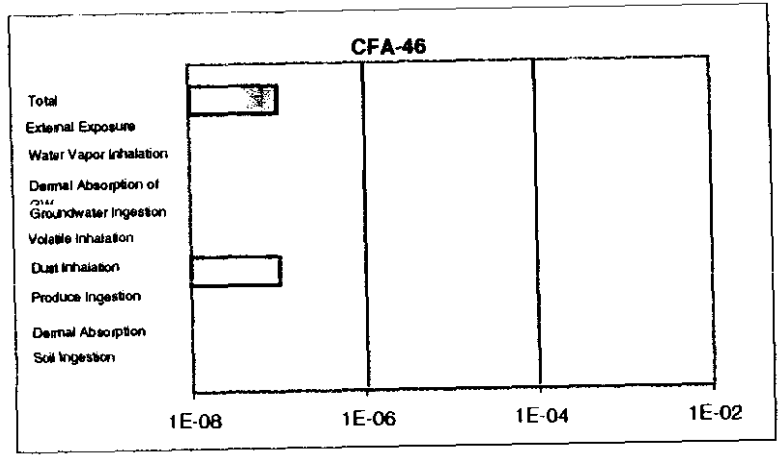


Figure 6-9. (continued).



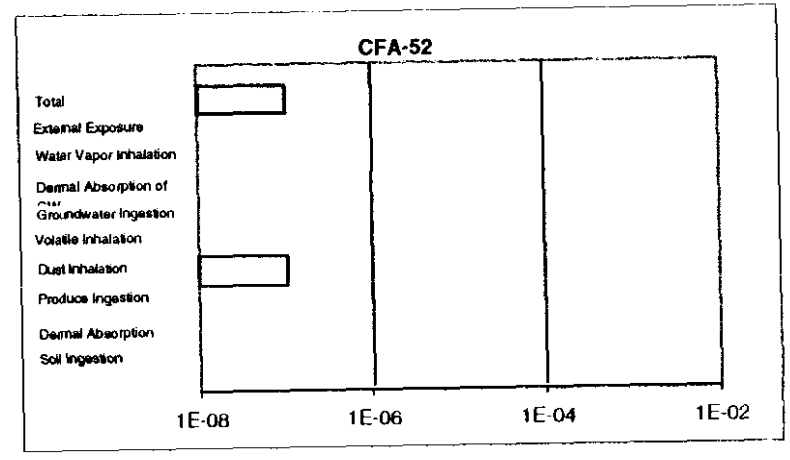


Figure 6-9. (continued).

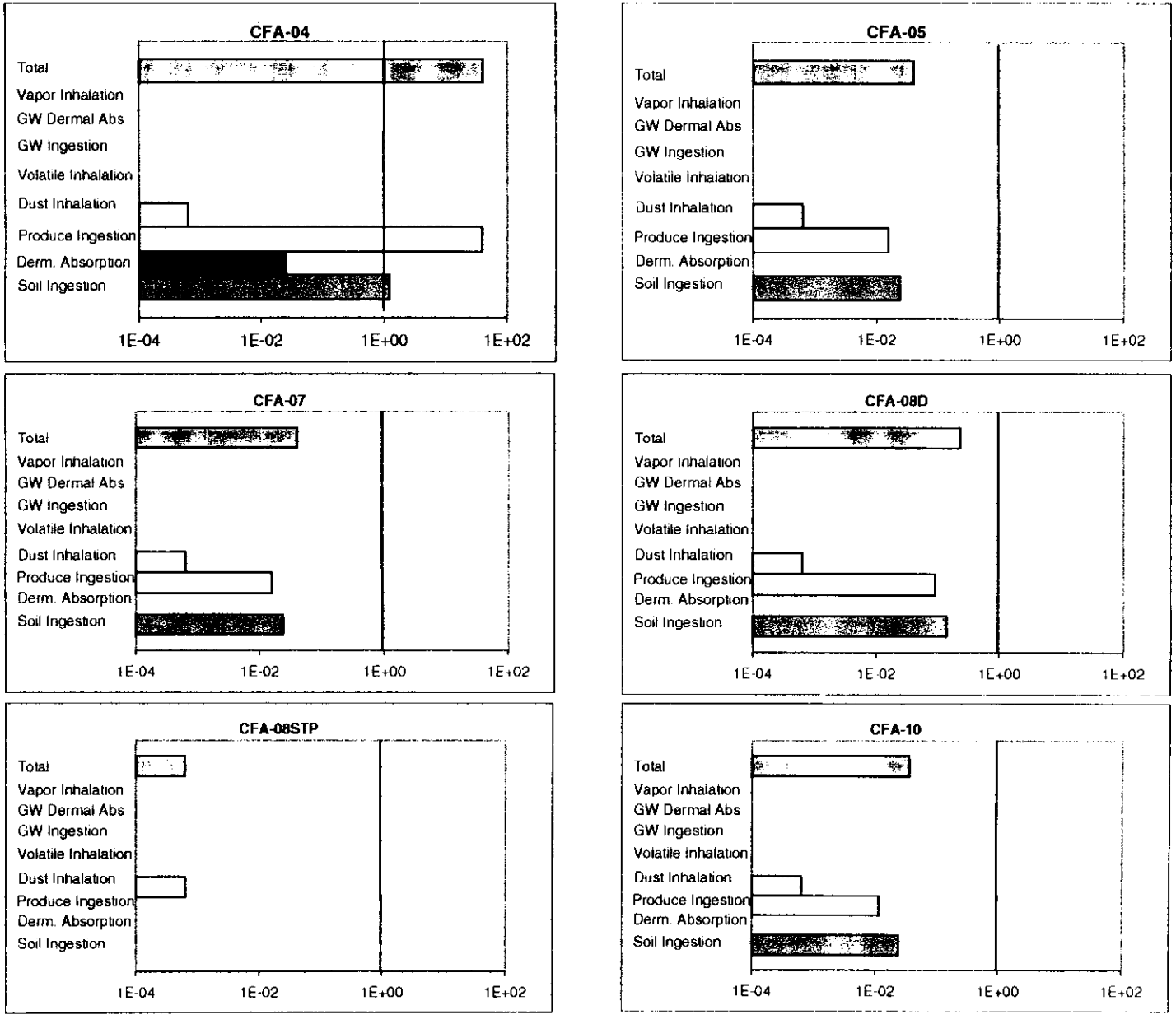
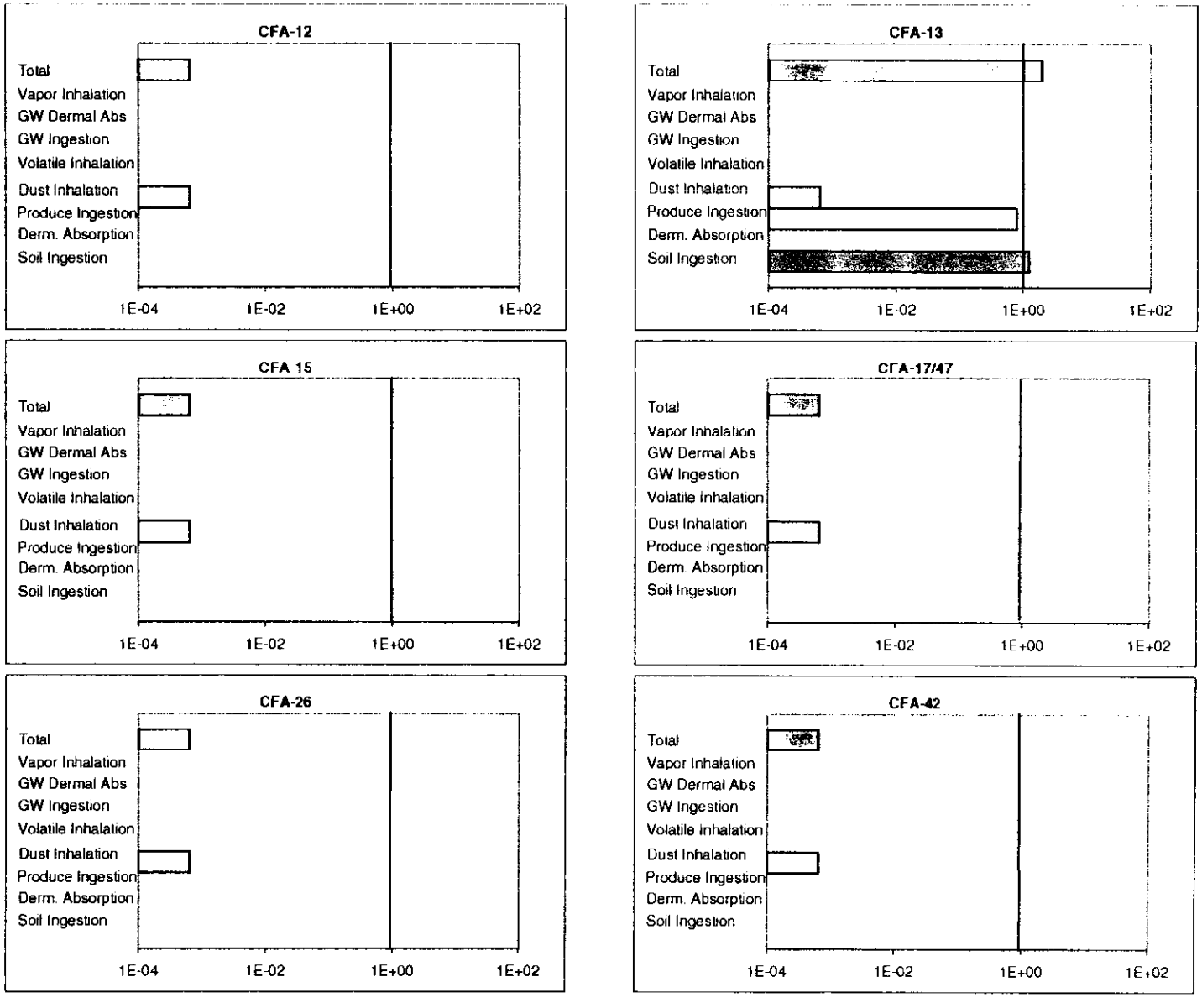


Figure 6-10. Total hazard indices for resident at 100 years.



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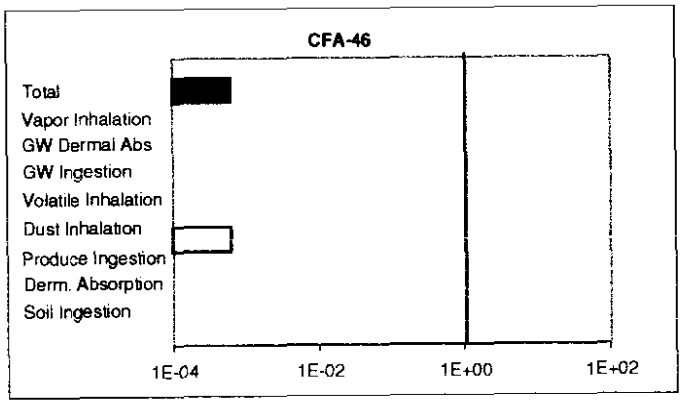

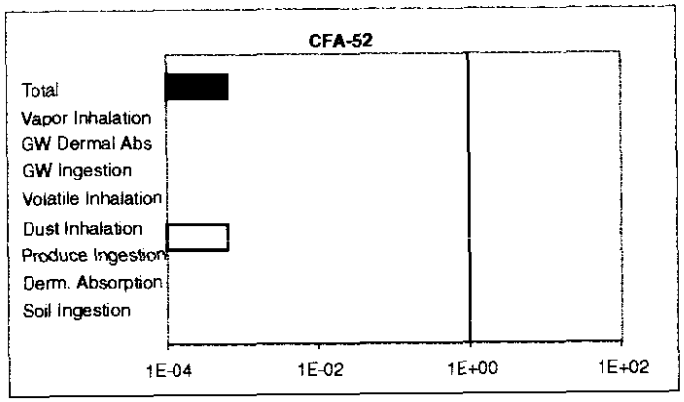

Figure 6-10. (continued).

Section 6.3.1.1, it is conservatively assumed that chemical degradation does not occur for nonradionuclides; as a result, the potential noncancer risks presented for the future occupational scenario are equivalent to those calculated for the current occupational scenario. The potential noncancer risks for these two receptors are therefore presented together. Potential risks could not be quantified for four of the COPCs identified for WAG 4 because EPA-verified toxicity values are not currently available. A qualitative risk characterization for these chemicals is presented in Section 6.5.3.

6.5.2.1.1 Cumulative Risk Results. The following sections present the results of the cumulative risk assessment for the WAG 4 air and groundwater exposure pathways. The risk results presented in this section are based on the WAG-wide risk results for the air exposure pathways (i.e., inhalation of fugitive dust, inhalation of volatiles) and the groundwater exposure pathways (i.e., ingestion of groundwater, dermal absorption of groundwater, inhalation of water vapor from indoor groundwater use).

6.5.2.1.2 Potential Cumulative Excess Cancer Risks: Current Occupational Scenario. The cumulative excess cancer risk for the current occupational worker from inhalation of airborne particulates is 6E-08. Pu-239, U-234, and U-238 contribute to the majority of the cumulative potential inhalation risk (40 percent, 20 percent, and 20 percent, respectively). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Volatile COPCs were not detected in surface soils (i.e., soils from 0 to 0.5 ft bgs); therefore, cumulative current risks are not assessed for inhalation of volatiles.

6.5.2.1.3 Potential Cumulative Excess Cancer Risks: Future Occupational Scenario. The cumulative excess cancer risk for the future occupational worker from inhalation of airborne particulates is 6E-08. Pu-239, U-234, and U-238 contribute to the majority of the cumulative potential inhalation risk (40 percent, 20 percent, and 20 percent, respectively). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Volatile COPCs were not detected in surface soils (i.e., soils from 0 to 0.5 ft bgs); therefore, cumulative future risks are not assessed for inhalation of volatiles.

6.5.2.1.4 Potential Cumulative Excess Cancer Risks: Future Residential Scenario. The cumulative excess cancer risk for the future resident from inhalation of airborne particulates is 1E-07. Arsenic, U-234, and U-238 contribute to the majority of the cumulative potential inhalation risk (55 percent, 16 percent, and 17 percent, respectively). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06.

The cumulative excess cancer risk from inhalation of volatiles could not be quantified because toxicity data are not currently available for the one volatile COPC that was detected in soils from 0 to 10 ft bgs (i.e., phenanthrene). A qualitative assessment of potential risks from exposure to phenanthrene is presented in Section 6.5.3.

The cumulative excess cancer risk from exposure to groundwater for the future residential scenario is 2E-09. As shown in Section 6.3.3.3, groundwater modeling results indicate that Eu-152, chlorodifluoromethane, and phenol are the only COPCs that expected to arrive at the hypothetical receptor well location at the 100- to 130-year exposure period. Of these COPCs, potential carcinogenic effects are only associated with Eu-152.

6.5.2.1.5 Potential Cumulative Noncancer Risks: Current and Future Occupational Scenario. The cumulative HI for the current occupational worker from inhalation of airborne particulates is 0.001. This HI is well below the EPA threshold HI of 1.0. Mercury contributes to

all of the HI estimate. Volatile COPCs were not detected in surface soils (i.e., soils from 0 to 0.5 ft bgs); therefore, cumulative noncancer risks are not assessed for inhalation of volatiles.

6.5.2.1.6 Potential Cumulative Noncancer Risks: Future Residential Scenario. The cumulative HI for the future resident from inhalation of airborne particulates is 1E-03. This HI is well below the EPA threshold HI of 1.0. Mercury contributes to all of the HI estimate. The total noncancer risk from inhalation of volatiles could not be quantified because no toxicity data are available for the one volatile COPC that was detected in soils from 0 to 10 ft bgs (i.e., phenanthrene). A qualitative assessment of potential noncancer risks from exposure to phenanthrene is presented in Section 6.5.3.

6.5.2.2 Site-specific Risk Results. The following sections present the site-specific risk results for WAG 4. The risk results presented for each site include risk results for site-specific exposure pathways (i.e., incidental soil ingestion, dermal contact with soil, ingestion of homegrown produce) and risk results for the WAG-wide exposure pathways (i.e., inhalation of particulates, inhalation of volatiles, groundwater ingestion, dermal contact with groundwater, and inhalation of water vapor from indoor groundwater use).

6.5.2.2.1 Potential Excess Cancer Risks: Current Occupational Scenario. Table D-43 presents the site-specific excess cancer risk estimates for the current occupational worker. Risk estimates are shown for each retained site and for each potentially complete exposure pathway identified for the current occupational worker. Potential risks estimated for this receptor at each retained site are discussed in the sections below.

6.5.2.2.1.1 CFA-04. The total excess cancer risk for the current occupational worker is 9E-06, or nine in one million (Table D-43). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Most of the total excess cancer risk at CFA-04 is associated with three exposure pathways: soil ingestion, dermal contact with soil, and external radiation exposure. These pathways contribute roughly 24, 13, and 63 percent, respectively, to the total risk estimate. Almost all of the risk associated with the two soil pathways is attributable to arsenic. Almost all of the risk associated with the external radiation exposure pathway is attributable to Cs-137.

As discussed in Sections 4.1.3 and 6.2.3, arsenic is not associated with known waste producing processes at WAG 4; however, arsenic is retained as a COPC for CFA-04 because the maximum detected concentration slightly exceeds the range of measured background concentrations at the INEEL. Past waste producing activities at CFA-04 may have resulted in concentrating naturally occurring levels of arsenic at this site; potential risks from arsenic estimated for this site are likely attributable to background levels.

6.5.2.2.1.2 CFA-05. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-05 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.3 CFA-07. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-07 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.4 CFA-08D. The total excess cancer risk for the current occupational worker is 2E-03, or two in one thousand (Table D-43). This risk level exceeds the EPA acceptable excess cancer

risk range of 1E-04 to 1E-06. Almost all (greater than 99 percent) of the risk estimated for CFA-08D is attributable to external radiation exposure to Cs-137 in soil. This risk estimate indicates that under the current exposure scenario (i.e., occupational), remediation is warranted for CFA-08D.

6.5.2.2.1.5 CFA-08STP. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-08STP is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.6 CFA-10. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-10 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.7 CFA-12. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-12 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.8 **CFA-13.** The total excess cancer risk for the current occupational worker is 9E-05, or nine in one hundred thousand (Table D-43). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Almost all (greater than 99 percent) of the risk estimated for CFA-13 is attributable to external radiation exposure to Ra-226 in soil.

6.5.2.2.1.9 ***CFA-15.*** The total excess cancer risk for the current occupational worker is 8E-05, or eight in one hundred thousand (Table D-43). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Almost all (greater than 99 percent) of the risk estimated for CFA-15 is attributable to external radiation exposure to Ra-226 in soil.

6.5.2.2.1.10 CFA-17/47. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-17/47 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.11 CFA-26. The total excess cancer risk for the current occupational worker is 9E-08, or nine in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-26 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.12 CFA-42. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-42 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.13 CFA-46. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-46 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.1.14 CFA-52. The total excess cancer risk for the current occupational worker is 6E-08, or six in one hundred million (Table D-43). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-52 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2 Potential Excess Cancer Risks: Future Occupational Scenario. Table D-45 presents the site-specific excess cancer risk estimates for the future occupational worker. Risk estimates are shown for each retained site and for each potentially complete exposure pathway identified for the current occupational worker. Potential risks estimated for this receptor at each retained site are discussed in the sections below.

6.5.2.2.2.1 CFA-04. The total excess cancer risk for the future occupational worker is 6E-06, or six in one million (Table D-45). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Most of the total excess cancer risk at CFA-04 is associated with three exposure pathways: soil ingestion, dermal contact with soil, and external radiation exposure. These pathways contribute roughly 46, 25, and 27 percent, respectively, to the total risk assessment. Almost all of the risk associated with the soil exposure pathways is attributable to arsenic. Over 50 percent of the risk associated with the external exposure pathway is attributable to U-238.

As discussed in Sections 4.1.3 and 6.2.3, arsenic is not associated with known waste producing processes at WAG 4; however, arsenic is retained as a COPC for CFA-04 because the maximum detected concentration slightly exceeds the range of measured background concentrations at the INEEL. Past waste producing activities at CFA-04 may have resulted in concentrating naturally occurring levels of arsenic at this site; potential risks from arsenic estimated for this site are likely attributable to background levels.

6.5.2.2.2.2 CFA-05. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-05 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.3 CFA-07. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-05 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.4 CFA-08D. The total excess cancer risk for the future occupational worker is 2E-04, or two in ten thousand (Table D-45). This risk level slightly exceeds the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Almost all of the risk (greater than 99 percent) estimated for CFA-08D is attributable to external radiation exposure to Cs-137 in soil. This risk estimate indicates that under the future occupational exposure scenario, remediation is warranted for CFA-08D.

6.5.2.2.2.5 CFA-08STP. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-08STP is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.6 CFA-10. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable

excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-10 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.7 CFA-12. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-12 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.8 CFA-13. The total excess cancer risk for the future occupational worker is 9E-05, or nine in one hundred thousand (Table D-45). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Almost all (greater than 99 percent) of the risk estimated for CFA-13 is attributable to external radiation exposure to Ra-226 in soil.

6.5.2.2.2.9 CFA-15. The total excess cancer risk for the future occupational worker is 9E-05, or nine in one hundred thousand (Table D-45). This risk level is well within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Almost all (greater than 99 percent) of the risk estimated for CFA-15 is attributable to external radiation exposure to Ra-226 in soil.

6.5.2.2.2.10 CFA-17/47. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-17/47 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.11 CFA-26. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-26 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.12 CFA-42. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-42 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.13 CFA-46. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-46 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.2.14 CFA-52. The total excess cancer risk for the future occupational worker is 6E-08, or six in one hundred million (Table D-45). This risk level is well below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The risk estimate for CFA-52 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3 Potential Excess Cancer Risks: Future Residential Scenario. Table D-47 presents the excess cancer risk estimates for the future residential scenario. Risk estimates are shown for each retained site and for each potentially complete exposure pathway identified for the future resident. Potential risks estimated for this receptor at each retained site are discussed in the sections below. The site-wide risk estimates for the groundwater exposure pathways (i.e., ingestion, dermal contact, inhalation of volatiles during) contribute minimally to the total risk estimate; together, these pathways contribute to less than 0.6 percent of the total cancer risk estimate for each site.

6.5.2.2.3.1 CFA-04. The total excess cancer risk for the future residential scenario is 4E-05, or four in one hundred thousand (Table D-47). This risk level is within the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. Approximately 65 percent of the total excess cancer risk at CFA-04 is associated with soil ingestion. Arsenic contributes to almost all of the estimated risks from soil ingestion. Dermal contact with soil, ingestion of homegrown produce, and external radiation exposure comprise most of the remainder of the total site risks (approximately 11, 7, and 17 percent, respectively).

As discussed in Sections 4.1.3 and 6.2.3, arsenic is not associated with known waste producing processes at WAG 4; however, arsenic is retained as a COPC for CFA-04 because the maximum detected concentration slightly exceeds the range of measured background concentrations at the INEEL. Past waste producing activities at CFA-04 may have resulted in concentrating naturally occurring levels of arsenic at this site; potential risks from arsenic estimated for this site are likely attributable to background levels.

6.5.2.2.3.2 CFA-05. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-05 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3.3 CFA-07. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-07 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3.4 CFA-08D. The total excess cancer risk for the future residential scenario is 4E-04, or four in ten thousand (Table D-47). This risk level exceeds the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-08D is attributable to external radiation exposure to Cs-137 in soil. This risk estimate indicates that under the future residential exposure scenario, remediation is warranted for CFA-08D.

6.5.2.2.3.5 CFA-08STP. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-08STP is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3.6 CFA-10. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-10 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3.7 CFA-12. The total excess cancer risk for the future residential scenario is 6E-04, or six in ten thousand (Table D-47). This risk level exceeds the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 98 percent) for CFA-12 is attributable to external radiation exposure to Cs-137 present in fractures of the basalt at a depth of 2.6 m (8.5 ft). This risk estimate indicates that under the future residential exposure scenario, remediation may be warranted for CFA-12. However, as described in Section 4 (Nature and Extent of Contamination), it is important to note that soils at CFA-12 have been remediated and that any residual contamination that exists at the site is in the basalt.

6.5.2.2.3.8 CFA-13. The total excess cancer risk for the future residential scenario is 6E-04, or six in ten thousand (Table D-47). This risk level exceeds the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 97 percent) for CFA-13 is attributable to external radiation exposure to Ra-226 in soil. This risk estimate indicates that under the future residential exposure scenario, additional remediation is warranted for CFA-13.

6.5.2.2.3.9 CFA-15. The total excess cancer risk for the future residential scenario is 4E-04, or four in ten thousand (Table D-47). This risk level exceeds the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-13 is attributable to external radiation exposure to Ra-226 in soil. This risk estimate indicates that under the future residential exposure scenario, additional remediation is warranted for CFA-15.

6.5.2.2.3.10 CFA-17/47. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-17/47 is based on the site-wide risk estimate for inhalation of particulates.

6.5.2.2.3.11 CFA-26. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-26 is based on the site-wide risk estimate for inhalation to particulates.

6.5.2.2.3.12 CFA-42. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-42 is based on the site-wide risk estimate for inhalation to particulates.

6.5.2.2.3.13 CFA-46. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-46 is based on the site-wide risk estimate for inhalation to particulates.

6.5.2.2.3.14 CFA-52. The total excess cancer risk for the future residential scenario is 1E-07, or one in ten million (Table D-47). This risk level is below the EPA acceptable excess cancer risk range of 1E-04 to 1E-06. The majority of the risk estimate (greater than 99 percent) for CFA-52 is based on the site-wide risk estimate for inhalation to particulates.

6.5.2.2.4 Potential Noncancer Risks: Current and Future Occupational Scenarios. Tables D-44 and D-46 present the noncancer risk estimates (i.e., hazard indices) for the current and future occupational workers. The noncancer risk estimates for these two potential receptors are identical because it is assumed that chemical degradation of nonradionuclides does not occur. Noncancer risk estimates are shown for each retained site and for each potentially complete exposure pathway identified for the current and future occupational workers.

6.5.2.2.4.1 CFA-04. The total estimated HI for CFA-04 for current and future occupational workers is 0.7 (Tables D-44 and D-46). This HI is below the EPA threshold HI of 1.0. Almost all of the estimated HI (greater than 98 percent) is associated with incidental ingestion of mercury in soil.

6.5.2.2.4.2 CFA-05. The total HI for CFA-05 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.3 CFA-07. The estimated HI for CFA-07 for the current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.4 CFA-08D. The estimated HI for CFA-08D for the current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.5 CFA-08 STP. The estimated HI for CFA-08STP for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.6 CFA-10. The estimated HI for CFA-10 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.7 CFA-12. The estimated HI for CFA-12 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.8 CFA-13. The estimated HI for CFA-13 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.9 CFA-17/47. The estimated HI for CFA-17/47 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.10 CFA-26. The estimated HI for CFA-26 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.11 CFA-42. The estimated HI for CFA-42 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.12 CFA-46. The estimated HI for CFA-46 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.4.13 CFA-52. The estimated HI for CFA-52 for current and future occupational workers is 0.001 (Tables D-44 and D-46). This HI is well below the EPA threshold HI of 1.0. The estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5 Potential Noncancer Risks: Future Residential Scenario. Table D-48 presents the noncancer risk estimates (i.e., hazard indices) for the future residential scenario. Noncancer

risk estimates are shown for each retained site and for each potentially complete exposure pathway identified for the future resident. Potential risks estimated for this receptor at each retained site are discussed in the sections below. The site-wide risk estimates for the groundwater exposure pathways (i.e., ingestion, dermal contact, inhalation of volatiles during) contribute minimally to the total risk estimate; together, these pathways contribute to less than 0.4 percent of the total risk estimate for each site.

6.5.2.2.5.1 CFA-04. The estimated HI for CFA-04 for the future residential scenario is 60 (Table D-48). This HI exceeds the EPA threshold HI of 1. The majority of the noncancer risk (approximately 97 percent) is associated with ingestion of homegrown produce; mercury contributes most significantly to the hazard quotient calculated for this exposure pathway. The mercury noncancer risk estimate for the homegrown produce pathway is largely based on the assumed soil-to-water partition coefficient (K_d); uncertainties associated with this parameter are discussed in Section 6, Uncertainty Analysis. Some of the noncancer risk (approximately two percent) is associated with soil ingestion; ingestion of mercury contributes most significantly to the HQ estimated for this exposure pathway.

6.5.2.2.5.2 CFA-05. The estimated HI for CFA-05 the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.3 CFA-07. The estimated HI for CFA-07 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.4 CFA-08D. The estimated HI for CFA-08D for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.5 CFA-08STP. The estimated HI for CFA-08STP for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.6 CFA-10. The estimated HI for CFA-10 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.7 CFA-12. The estimated HI for CFA-12 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.8 CFA-13. The estimated HI for CFA-13 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.9 CFA-15. The estimated HI for CFA-15 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.10 CFA-17/47. The estimated HI for CFA-17/47 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.11 CFA-26. The estimated HI for CFA-26 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.12 CFA-42. The estimated HI for CFA-42 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.13 CFA-46. The estimated HI for CFA-46 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.2.2.5.14 CFA-52. The estimated HI for CFA-52 for the future residential scenario is 0.001 (Table D-48). This HI is well below the EPA threshold HI of 1.0. Over 99 percent of the estimated HI is based on the site-wide HI for inhalation of particulates.

6.5.3 Risk Characterization for COPCs without Toxicity Values

EPA-verified toxicity values are not currently available for three of the COPCs identified for WAG 4 [i.e., benzo(g,h,i)perylene, lead, TPH]. For these COPCs, EPA (1989a) recommends a qualitative, rather than quantitative, evaluation of potential risks. These evaluations are presented below.

Benzo(g,h,i)perylene is a polycyclic aromatic hydrocarbon (PAHs) that was identified as a soil COPC at CFA-13 and CFA-17/47. Benzo(g,h,i)pyrelene does not have any available toxicity data, but the toxicity of contaminants in the PAH family are usually estimated by comparison against the toxicity of benzo(a)pyrene.

Benzo(a)pyrene has been thoroughly studied by the medical community and it has been shown to be a Class B2 carcinogen (i.e., it is a probable human carcinogen). In contrast, there is no evidence from animal toxicity studies that benzo(g,h,i)pyrelene produces any carcinogenic health effects.

The EPA Region III 1E-06 risk-based concentration for benzo(a)pyrene is 0.088 mg/kg, and the 0-10 ft average concentration of benzo(g,h,i)pyrelene at CFA-13 is estimated to be 1.79 mg/kg. The CFA-13 average concentration is 20 times greater than the benzo(a)pyrene risk-based concentration, so if benzo(g,h,i)pyrelene were exactly as toxic as benzo(a)pyrene, its risk would be approximately equal to 2E-05 at CFA-13. Similarly, the 0-10 ft average concentration for benzo(g,h,i)perylene at CFA-17/47 is estimated to be 1.1E-02 mg/kg, so the risk for the contaminant would be equal to 1E-07 if it were as toxic as benzo(a)pyrene. These risk results are upper bound estimates since benzo(g,h,i)pyrelene has been shown to be much less toxic than benzo(a)pyrene.

As shown in Table D-47, the total calculated risk at CFA-13 is 6E-04, and the total risk at CFA-17/47 is 1E-07. As a result, benzo(g,h,i)perylene would not significantly change the risk estimates at either site, even if it were as toxic as benzo(a)pyrene.

6.5.3.1 Lead. Lead is identified as a soil COPC at CFA-10 and CFA-13. Exposure point concentrations for soil lead at CFA-10 and CFA-13 were compared against the EPA recommended

400 mg/kg screening level concentration for lead in residential soil at CERCLA and RCRA Corrective Action sites (OSWER Directive #9355.4-12, EPA 1994b). The 400 mg/kg concentration derived by EPA is based on the pharmacokinetic modeled response (using the Integrated Exposure Uptake Biokinetic [IEUBK] model) of a hypothetical child to lead exposures, based on default parameters. It represents a level below which no further action or study is warranted, provided no special circumstances (e.g., presence of wetlands) warrant further study. The 400 mg/kg concentration is associated with an expected response of a hypothetical child to lead exposure via soil and dust ingestion, and is intended to limit exposure such that the hypothetical child or group of similarly exposed children would have an estimated risk of no more than five percent exceeding the U.S. Centers for Disease Control 10 ug/dL blood lead level of concern (EPA 1994b).

The calculated lead exposure point concentration at CFA-10 for the occupational and residential exposure scenarios are 3,300 mg/kg and 165 mg/kg, respectively. The residential exposure point concentration does not exceed the UESPA (1994b) residential soil lead screening level, indicating that soil lead concentrations at CFA-10 are not expected to pose an unacceptable health risk to children (or adults) under a long-term residential exposure scenario. The occupational lead exposure point concentration at CFA-10 was also compared to the residential screening level because an occupational screening level is not available currently. The occupational lead exposure point concentration exceeds the residential screening level by a factor of eight, indicating that lead levels at CFA-10 may be of concern for occupational workers and future residents who receive exposures from only shallow surface soils.

The calculated lead exposure point concentration at CFA-13 is 261 mg/kg (residential scenario only). The residential exposure point concentration does not exceed the USEPA (1994b) residential soil lead screening level, indicating that soil lead concentrations at CFA-13 are not expected to pose an unacceptable health risk to children (or adults) under a long-term residential exposure scenario.

6.5.3.2 TPH. TPH-d is identified as a COPC at CFA-26, CFA-46, and CFA-52. Potential risks from TPH-d via soil exposure pathways were not assessed in the BRA because detections of TPH-d occur at depths in excess of 3.05 m (10 ft) bgs. TPH-d was, however, retained for the WAG-wide groundwater evaluation. Because of the lack of EPA-approved toxicity values, TPH-d was not included in the risk calculations. However, a comparison can be made of predicted TPH groundwater concentrations to the IDEQ groundwater cleanup levels for TPH. TPH-heating oil is also evaluated in this comparison because this TPH mixture has been reported at CFA-26 (see Section 6.3.3.3). The predicted cumulative groundwater peak concentrations of TPH-diesel and TPH-heating oil are well below the IDEQ groundwater cleanup level of 100 mg/L (see comparison below). The predicted time and concentrations for TPH to reach peak concentrations ranges from 427 to 464 years (see Section 6.3.3.3) at concentrations of 1.1 to 0.00115 mg/L, respectively.

6.6 Uncertainty Analysis

The risk assessment results presented in this BRA are very dependent on the methodologies described in Section 6.3. These analysis methods were developed over a period of several years by INEEL risk management and risk assessment professionals to provide realistic, and yet conservative, estimates of human health risks at WAG 4. Nonetheless, if different risk assessment methods had been used, the BRA would likely have produced different risk assessment results. To ensure that the risk estimates are conservative, health protective assumptions that tend to envelope the plausible upper limits of human health risks are used throughout the BRA. Therefore, risk estimates that may be calculated by other risk assessment methods are not likely to be significantly higher than the estimates presented in Section 6.5.

The BRA results in Section 6.5 are useful for evaluating which WAG 4 release sites require remediation because the results are calculated in a consistent manner. This consistency allows for direct comparison of the risk assessment results for a given release site with the results for every other site included in the evaluation. Changes in a given assumption used in the evaluation would, in general, produce similar changes in the risk results for all of the release sites evaluated. As described in the remainder of this section, the BRA results include inherent uncertainty, but despite this uncertainty, the consistency of the analysis makes the results useful for making remediation decisions.

Uncertainty in this BRA is produced by uncertainty factors in the following four stages of analysis:

1. Data collection and evaluation
2. Exposure assessment
3. Toxicity assessment
4. Risk characterization.

The following subsections discuss each of these four stages in more detail, and

6.6.1 Data Collection and Evaluation Uncertainties

Uncertainties associated with data collection and evaluation are produced by variability in observed concentrations caused by sampling design and implementation, laboratory analysis methods, seasonality, contaminant level variation, and natural concentration variation. Optimizing the usability of sampling data involves quantifying these uncertainties.

The effect of uncertainty introduced from sample collection and analysis is reduced by basing risk estimates on the 95% UCL of the mean for the WAG 4 COPC concentration estimates. The resulting concentration estimates, used to estimate intakes, are an upper bound estimate of the concentrations observed at the retained sites. This approach is health protective and accounts for the uncertainty introduced by sampling, analysis, seasonality, and natural variation.

A major assumption included in the BRA analysis is that all significant sources of contamination at WAG 4 have been identified and sampled. If a source of contamination has not been identified and sampled, the risks from the contamination are not included in the BRA.

CFA-12 includes an example of contamination that may not have been detected in the site's sampling activities. Table 4-1 of the OU 4-09 Track 2 Summary Report (Gianotto et al., 1996) shows that the 1993 sampling of the CFA-12 drain sediments produced maximum detections of cadmium, calcium, mercury, and lead that exceeded INEEL background concentrations. At the time the drains were removed, these relatively low concentrations were not considered to be significant, so the samples that were collected after the removal were not tested for metals. All of the metal contamination that was detected in the 1993 sampling was removed with the drains, but there is a small chance that low levels of undetected metal contamination still exists in the basalt beneath the drains.

One of the first steps in the BRA was a screening of release sites and contaminants (see Section 6.2). The purpose of this screening activity was to help focus the BRA on sites and contaminants that are likely to produce adverse human health effects. The screening process was designed to be conservative so that all sites and contaminants that have a reasonable potential for causing adverse human

health effects would pass the screening, and therefore would be evaluated in the BRA. If in fact the screening process was not conservative enough, and sites or contaminants that could cause adverse human health effects were inappropriately screened out, then the BRA risk results presented in Section 6.5 would be underestimated. A contamination source would have to be small to be inappropriately screened, so any underestimation of risk would be slight if a site or contaminant were inappropriately screened.

Tentatively identified compounds (TICs) were detected at several of the WAG 4 release sites. These compounds were not included in the BRA risk calculations. In accordance with EPA risk assessment guidance (EPA 1989a), the TICs were omitted because they were not detected frequently and because the compounds are not expected to have been released.

All of the release sites evaluated in the BRA have varying levels of uncertainty associated with the contaminant concentrations evaluated in the BRA. Additionally, all of the evaluated concentrations were estimated using conservative assumptions about the nature and extent of contamination at the various release sites. The concentration term uncertainties and conservative assumptions are summarized in Table 6-12.

6.6.2 Exposure Assessment

Uncertainties associated with the exposure assessment are produced by characterizing transport, dispersion, and transformation of COPCs in the environment; establishing exposure settings; and deriving estimates of chronic intake. The initial characterization that defines the exposure setting for a site involves many professional judgments and assumptions. Definition of the physical setting, population characteristics, and selection of the chemicals included in the risk assessment are examples of areas for which a quantitative estimate of uncertainty cannot be achieved because of the inherent reliance on professional judgment. Assumptions and supporting rationale regarding these types of parameters, along with the potential impact on the uncertainty (i.e., overestimation or underestimation of uncertainty), are included in Table 6-12.

An aspect of the risk assessment that tends to exaggerate risk results is the evaluation of contaminants with background concentrations that produce calculated risks in excess of 1E–06. An example of this type of contaminant is arsenic. This metal is commonly detected in INEEL soils at concentrations that are slightly higher than the accepted background concentration. Arsenic, however, is not associated with known waste producing processes at WAG 4. Detected concentrations of arsenic are likely to be attributable to background concentrations; detected concentrations that slightly exceed accepted background concentrations are likely due to variations in background levels from site to site. For this reason, and because the toxicity values for arsenic are conservative (i.e., high slope factor, low reference dose), risks are likely to be overestimated at those sites at which arsenic was retained as a COPC.

As discussed in Section 4, the contaminant source terms evaluated in the BRA were calculated by "volume weighting" measured site concentrations. Volume weighting refers to the process of measuring contaminant concentrations at various locations and depths at a given release site, estimating the volume of soil that is represented by a given measurement or group of measurements, and deriving average contaminant concentrations at the site by weighting the measurements with the associated soil volumes. This process produces reasonable estimates of a site's average contaminant concentrations as long as the site was thoroughly sampled. If the contamination at a given site was not well defined, volume weighting could produce errors in the site's average concentrations. These errors could either over or under estimate the true average contaminant concentrations at the site, depending on the results of the site's sampling

investigation. Details of the sampling investigations evaluated in the RI/FS are discussed in Section 4, and summarized in Table 6-12.

The only contaminant loss mechanism considered in the BRA is radioactive decay. Other loss mechanisms such as leaching, wind erosion, etc., are assumed to be negligible. The reason for this assumption is that environmental sampling has shown that most contaminants do not migrate from most INEEL release sites. As a result of this observation, very few studies have been performed to evaluate these mechanisms, so there is very little site-specific information available to estimate the exact effects of these removal mechanisms.

Omitting removal mechanisms other than radioactive decay tends to overestimate risk for all exposure routes because it leads to assuming a given mass of contaminant will cause exposures to multiple exposure routes. For example, leaching is omitted in the soil pathway analysis even though leaching is the mechanism that produces the contamination evaluated in the groundwater pathway analysis. As a result of the omission, a given mass of contamination can affect both the soil pathway and groundwater pathway risk results. Upper bound infiltration and contaminant leachability assumptions are used in the groundwater pathway analysis to estimate future groundwater contaminant concentrations. Applying these same upper bound assumptions to the soil pathway analysis would likely produce an underestimation of soil pathway risks. To avoid this possibility, leaching is omitted from the soil pathway analysis, so that upper bound risk results are calculated for both the soil pathway and groundwater pathway exposure routes.

The estimated residential exposure (i.e., chemical intake) to mercury via the homegrown produce ingestion exposure pathway contributes to over 90 percent of the estimated HI for CFA-04 which includes the pond area, the mercury retort equipment staging area, and the windblown area The estimated intake of mercury from this exposure pathway is largely driven by the assumed value of 100 for the K_d (soil-to-water partition coefficient). The assumed value is based on the DOE (1994) suggested K_d values. The suggested value of 100 is conservative; other K_d values in the literature are shown to be as much as an order of magnitude lower (Baes et al. 1984). Reduction of the K_d by an order of magnitude can result in reduction of the estimated homegrown produce pathway HI by a factor of 6.

One of the purposes of the BRA is to estimate upper bound risks from WAG 4 contaminant releases based on best available site specific information. Omitting removal mechanisms that have not been studied on a site specific basis, and which are likely to produce only small errors in the calculated risk results, is consistent with this objective.

The sites containing radionuclide contamination were examined for on-site risk from external radiation exposure. However, external radiation exposure from gamma emitting radionuclides may extend outward from a site boundary if the radiation is attenuated only by air, so external radiation dose may be additive if a receptor is in the proximity of several sites containing radionuclide soil contamination. Risk results for the external radiation pathway (see Tables D-43, - 45, and -47) indicate that potential risks from external radiation exceed 1E-04 to 1E-06 EPA acceptable excess cancer risk range for only one of the eleven quantitatively evaluated for risk. Previous external radiation risk estimates from other INEEL sites with radionuclide activities greater than those measured at WAG 4 have been insignificant. Therefore, external radiation risks from doses that may be received outside individual site boundaries are likely to be insignificant and were not evaluated.

Table 6-12. Summary of Source Term Uncertainties for the OU 4-13 BRA.

Release Sites		Source Term Uncertainties and/or Assumptions
CFA-13	Dry Well (South of CFA-640)	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Of the 19 calculated site-specific exposure point concentrations, all are based on the maximum detected concentration. The area of contamination is assumed to exist uniformly across the site, even though only two of the nine COPCs were detected in 100% of the site-wide samples. The other COPCs were detected in at least 14.3% of the samples. Contamination is assumed to exist down to 9.1 m (30 ft), even though positive detections of chemicals in the vadose zone are reported only to a depth of 6.1 m (20 ft). The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-13 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-13 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-15	Dry Well (CFA-674)	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Of the three calculated site-specific exposure point concentrations, all are based on the maximum detected concentration. The one identified COPC was detected in 100% of the site-wide samples. The area of contamination is assumed to exist uniformly across the site. Contamination is assumed to exist down to 8 m (26 ft), even though positive detections of chemicals in the vadose zone are reported only to a depth of 4.9 m (16 ft). The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-15 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-15 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-04	Pond (CFA-674)	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Of the 18 calculated site-specific exposure point concentrations, six are based on the maximum detected concentration. The area of contamination is assumed to exist uniformly across the site, even though only two of the six COPCs were detected in 100% of the site-wide samples. The other COPCs were detected in at least 48.0% of the samples. The area of contamination is assumed to exist uniformly across the site. Contamination is assumed to exist down to 5.5 m (18 ft), even though positive detections of chemicals in the vadose zone are reported only to a depth of 2.4 m (8 ft). The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-04 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-04 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.

Table 6-12. (continued).

Release Sites		Source Term Uncertainties and/or Assumptions
CFA-17/47	Fire Department Training Area (bermed) and Fire Station Chemical Disposal	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. The area of contamination is assumed to exist uniformly across the site, even though none of the COPCs was detected in more than 5% of the site-wide samples. Contamination is assumed to exist down to 4 m [13 ft], even though positive detections of chemicals in the vadose zone are reported only to a depth of 0.9 m (3 ft). Sampling depths did occur at 7 m (23 ft) bgs, but results from the 0.9 to 7 m (3 ft to 23 ft) bgs depth interval did not indicate the presence of COPCs. The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-17/47 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-17/47 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-07	French Drains E/S (CFA-633)	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. All of the four calculated site-specific exposure point concentrations are based on the maximum detected concentration. The area of contamination is assumed exist uniformly across the site (both drains), even though only three of the four COPCs were detected in 100% of the number of site-wide samples. The other COPC was detected in 66.7 % of the site-wide samples. Contamination is assumed to exist down to 7.2 m [23.5 ft], even though positive detections of chemicals in the vadose zone are reported only to a depth of 4.1 m (13.5 ft). The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-07 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-07 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-12	French Drains (2) (CFA-690) [South Drain only]	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Of the seven calculated site-specific exposure point concentrations, all are based on the maximum detected concentration. The area of contamination is assumed to exist uniformly across the south drain. Contamination is present in a basalt fracture at a depth of 2.6 m (8.5 ft). The depth to basalt is assumed to occur at 2.4 m (8 ft). Soils at this site have been excavated and backfilled with clean fill; residual contamination is in the basalt. Inclusion of this site for quantitative evaluation in the BRA is conservative because the soil has already been remediated. It is assumed that COPCs are contained within the soil above the 2.6 m (8.5 ft) level. It is also assumed that COPCs may occur from 0 to 2.6 m (0 to 8.5 ft) for the future residential scenario even though residual contamination is likely to remain immobile in the basalt. These assumptions may cause the calculated risks at the site to be overestimated.

Table 6-12. (continued).

Release Sites		Source Term Uncertainties and/or Assumptions
CFA-08	Drainfield	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Of the nine calculated site-specific exposure point concentrations, seven are based on the maximum detected concentration. The area of contamination is assumed to exist uniformly across the drainfield, even though site-wide detection frequencies for each of the three COPCs are no greater than 72.3%. Contamination is assumed to exist at 10 m (32 ft) bgs. The depth to basalt is assumed to occur at 10 m (32 ft). It is assumed that COPCs will not migrate downward beyond 10 m (32 ft) due to the presence of basalt at 10 m (32 ft). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-08	Sewage Treatment Plant (CFA-691)	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. Both of the two calculated site-specific exposure point concentrations are based on the 95% UCL. The area of contamination is assumed to exist uniformly across the sewage treatment plant, even though site-wide detection frequencies for the two COPCs are 73.1% and 100%. Contamination is assumed to exist down to 11.3 m [37.25 ft], even though positive detections of chemicals in the vadose zone are reported only to a depth of 8.3 m (27.25 ft). The depth of contamination is based on the assumption that mobility of chemicals suspended in liquids in the vadose zone (i.e., waste water) at CFA-08 is 3 m (10 ft). This assumption is made to ensure that potential risks from exposures at CFA-08 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-10	Transformer Yard Oil Spills	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. The one calculated site-specific exposure point concentration for this site is based on the maximum detected concentration. The area of contamination is the area of the site based on process knowledge that there was no specific pattern of waste disposal. The maximum depth of contamination is 0.15 m (0.5 ft) bgs based on depths of measured concentrations. For purposes of evaluating residential exposure pathways, contamination from 0 to 3.05 m (0 to 10 ft) soil interval is assumed. This assumption is made to ensure that potential risks from exposures at CFA-10 are not underestimated (Section 6). These assumptions may cause the calculated risks at the site to be overestimated.
CFA-26	CFA-760 Pump Station Fuel Spill	CFA-26 was retained for groundwater modeling purposes. It is assumed that contamination is uniformly distributed across the site. TPH was detected in 100% of the number of samples at a depth interval of 1.5 to 3.4 m (5 to 11.25 ft) bgs for CFA-26. Basalt was encountered at a depth range of 2.9 to 3.4 m (9.5 to 11.25 ft) bgs. These assumptions may cause the calculated risks at the site to be overestimated due to the possibility that the COPCs in groundwater may not reach receptors.

Table 6-12. (continued).

Release Sites		Source Term Uncertainties and/or Assumptions
CFA-42	Tank Farm Pump Station Spills	Exposure point concentrations used for depth interval and volume-weighted concentrations are based on the 95% UCL or maximum detected concentration, whichever is less, instead of average (arithmetic mean) concentrations. The only calculated site-specific exposure point concentration is based on the 95% UCL. The area of contamination is assumed to exist uniformly across the site, even though the COPC detection frequency is less than 5%. Contamination is assumed to exist down to 6.1 m [20 ft]. The depth to basalt is assumed to occur at 6.1 m (20 ft). Soils at this site have been excavated and backfilled with clean fill; residual contamination is in the basalt. Inclusion of this site for quantitative evaluation in the BRA is conservative because the soil has already been remediated.
CFA-46	Cafeteria Oil Tank Spill (CFA-721)	CFA-46 was retained for groundwater modeling purposes. It is assumed that contamination is uniformly distributed across the site. Of the five Groundwater COPCs evaluated, only one was detected in 100% of the number of samples, while the other COPCs were detected in 71.4% of the number of samples. Contamination is assumed to exist at 4.9 m (16 ft) bgs. Basalt was encountered at 4.9 m (16 ft) bgs. These assumptions may cause the calculated risks at the site to be overestimated due to the possibility that the COPCs in groundwater may not reach receptors.
CFA-05	Motor Pool Pond	CFA-05 was retained for groundwater modeling purposes. It is assumed that contamination is uniformly distributed across the site. Contamination was detected from 0 to 5.8 m (0 to 19 ft) bgs. Of the 10 groundwater COPCs, nine were detected in 100% of the number of site-wide samples and the remaining COPC was detected in 86.5% of the number of site-wide samples. Basalt was encountered at 5.8 m (19 ft) bgs. These assumptions may cause the calculated risks at the site to be overestimated due to the possibility that the COPCs in groundwater may not reach receptors.
CFA-52	Diesel Fuel UST (CFA-730) at Bldg CFA-613 Bunkhouse	CFA-52 was retained for groundwater modeling purposes. It is assumed that contamination is uniformly distributed across the site. TPH was detected in 100% of the number of samples at a depth interval of 4.6 to 5 m (15 to 16.5 ft) bgs. Basalt was encountered at 4.9 m (16 ft) bgs. These assumptions may cause the calculated risks at the site to be overestimated due to the possibility that the COPCs in groundwater may not reach receptors.

6.6.3 Toxicity Assessment

Several important measures of toxicity are needed to conduct an assessment of risk to human health. RfDs are applied to the oral and inhalation exposure to evaluate noncarcinogenic and developmental effects, and SFs are applied to the oral and inhalation exposures to carcinogens. RfDs are derived from NOAELs or LOAELs and the application of uncertainty factors (UFs) and modifying factors (MFs). UFs are used to account for the variation in sensitivity of human subpopulations and the uncertainty inherent in extrapolation of the results of animal studies to humans, while MFs account for additional uncertainties in the studies used to derive the NOAEL or LOAEL. Uncertainty associated with SFs is accounted for by an assigned weight-of-evidence rating that reflects the likelihood that the toxicant is a human carcinogen. Weight-of-evidence classifications are tabulated and included in Table D-42, while a discussion of the UFs and MFs used to derive RfDs are presented in Section 6.4.

6.6.4 Risk Characterization

The last step in the risk assessment is risk characterization. As discussed in Section 6.5, risk characterization is the process of integrating the results of the exposure and toxicity assessments. The uncertainties defined throughout the analysis process are combined and presented as part of the risk characterization to provide an understanding of the overall uncertainty in the estimate of risk. Table 6-13 presents this qualitative assessment of uncertainty. See Section 8 for a summary of WAG 4 risks.

6.6.5 Sensitivity Analysis

A sensitivity analysis was performed to assess potential difference in risk estimates given changes to the exposure assumptions used in the human health assessment for OU 4-13. The objective of the analysis was to illustrate the magnitude of risk reduction achieved by varying values for selected exposure parameters (e.g., varying the exposure duration). To meet this objective, three baseline assumptions for a hypothetical future resident were modified:

1. Keeping the exposure point concentrations constant, the exposure parameters were modified to reflect probable and more realistic future resident exposures

2. Keeping the exposure point concentrations constant, the exposure parameters were modified to reflect conditions for a modified (i.e., not full time) resident

3. Keeping the exposure assessment assumptions constant, alter the exposure point concentrations to reflect average rather than upper bound conditions.

The following sections discuss the assumptions used in the sensitivity analysis and the observed impacts to the risk estimates.

6.6.5.1 Exposure Parameter Sensitivity. Table 6-14 illustrates the exposure assessment parameters that were altered for the future resident and the modified resident in the sensitivity analysis. Generally, the parameters used for the future resident (averaging time, exposure duration, exposure frequency, exposure time and ingestion rate) reflect more typical residential exposures than the parameter values used in the BRA base case analysis (see Section 6.5). The only parameters altered for the modified resident were exposure frequency and exposure time. Parameters not shown here are assumed to be consistent with those used in the BRA base case analysis.

Table 6-13. BRA Human Health Assessment Uncertainty Factors.

Uncertainty factor	Effect of uncertainty	Comments and Assumptions
Source term assumptions	May overestimate risk	All contaminants are assumed to be completely available for transportation away from the source zone. In reality, some contaminants may be chemically or physically bound to the source zone and unavailable for transport.
Natural infiltration rate	May overestimate risk	A conservative value of 10 cm/year was used for this parameter.
Moisture content	May overestimate or underestimate risk	Soil moisture contents vary seasonally in the upper vadose zone and may be subject to measurement error.
Water table fluctuations	May slightly overestimate or underestimate risk	The average value used is expected to be representative of the depth over the 30-year exposure period.
Mass of contaminants in soils is estimated by assuming a uniform contamination concentration in the source zone.	May overestimate or underestimate risk	There is a possibility that most of the mass of a contaminant at a site may exist in a hotspot that was not detected by sampling. If this condition existed, the mass of the contaminant used in the analysis might be underestimated. However, 95% UCLs or maximum detected contamination were used for all mass calculations, and these concentrations are assumed to exist at every point in each waste site; therefore, the mass of contaminants used in the analysis is probably overestimated.
Plug flow assumption in groundwater transport	Could overestimate or underestimate risk	Plug flow groundwater models will likely estimate a greater mass of contaminants will be transported to the aquifer than would occur under natural conditions, with respect to concentrations because dispersion is neglected, and mass fluxes from the source to the aquifer differ only by the time delay in the unsaturated zone (the magnitude of the flux remains unchanged). For nonradiological contaminants, the plug flow assumption is conservative because dispersion as completed in the models is not allowed to dilute the contaminant groundwater concentrations. For radionuclides, the plug flow assumption may or may not be conservative. Based on actual travel time, the radionuclide groundwater concentrations could be overestimated or underestimated because a longer travel time allows for more decay. If the concentration decrease because the travel time delay is larger than the neglected dilution from dispersion, the model will not be conservative.
All infiltration into WAG 4 is assumed to occur through the contaminated sites	Will overestimate risk	Infiltration that normally occurs between contaminated sites is assumed to be concentrated on contaminated sites. This assumption results on a probable overestimate of risk because more water is available in the model calculation to carry contaminants to the aquifer.
No migration of contaminants from the soil source prior to 1994	Could overestimate or underestimate risk	The effect of not modeling contaminant migration from the soil before 1994 is dependent on the contaminant half-life, radioactive in growth, and mobility characteristics.
Contaminant source terms assumed to be lognormally distributed	Could overestimate risk	If sampling data at a given site fits a normal distribution rather than a lognormal distribution, the 95% UCL of the near concentrations calculated for the site could be as much as 50% too high. EPA from Superfund sites are lognormally distributed (EPA 1992).
Chemical form assumptions	Could overestimate or underestimate risk	In general, the methods and inputs used in contaminant migration calculations, including assumptions made about the chemical forms of contaminants were chosen to err on the protective side. All contaminant concentration and mass are assumed available for transport. This assumption results in a probable overestimate of risk.

Table 6-13. (continued).

Uncertainty factor	Effect of uncertainty	Comments and Assumptions
Exposure scenario assumptions	May overestimate risk	The likelihood of future scenarios has been qualitatively evaluated as follows: resident - improbable industrial - credible. The likelihood of future onsite residential development is small. If future residential use of this site does not occur, then the risk estimates calculated for future onsite residents are likely to overestimate the true risk associated with future use of this site.
Exposure parameter assumptions	May overestimate risk	Assumptions regarding media intake, population characteristics, and exposure patterns may not characterize actual exposures.
Receptor locations	May overestimate risk	Groundwater ingestion risks are calculated for a point at the downgradient edge of an equivalent rectangular area. The groundwater risk at this point is assumed to be the risk from groundwater ingestion at every point within the WAG 4 boundaries. Changing the receptor location will affect only the risks calculated for the groundwater pathway because all other risks are site-specific or assumed constant at every point within the WAG 4 boundaries.
For the groundwater pathway analysis, all contaminants are assumed to be homogeneously distributed in a large mass of soil.	May overestimate or underestimate risk	The total mass of each COPC is assumed to be homogeneously distributed in the soil volume beneath the WAG 4 retained sites. This assumption tends to maximize the estimated groundwater concentrations produced by the contaminant inventories because homogeneously distributed contaminants would not have to travel far to reach a groundwater well drilled anywhere within the WAG 4 boundary. However, groundwater concentrations may be underestimated for a large mass of contamination located in a small area with a groundwater well drilled directly downgradient.
The entire inventory of each contaminant is assumed to be available for transport along each pathway	May overestimate risk	Only a portion of each contaminant's inventory is actually transported by each pathway.
Exposure duration	May overestimate risk	The assumption that an individual will work or reside at a contaminated site for 25 or 30 years is conservative. Short-term exposures involve comparison to subchronic toxicity values, which are generally less restrictive than chronic values.
Noncontaminant-specific constants (not dependent on contaminant properties)	May overestimate risk	Conservative or upper limit values were used for all parameters incorporated into intake calculations.
Exclusion of some hypothetical pathways from the exposure scenarios	May underestimate risk	Exposure pathways are considered for each scenario and eliminated only if the pathway is either incomplete or negligible compared to other evaluated pathways.
Poorly defined dermal absorption factors (ABS) values for most WAG 4 contaminants	May underestimate risk	A lack of ABS values for most WAG 4 contaminants may mean that dermal absorption risks are higher than expected. The possibility of unacceptable dermal absorption from soil risks being produced by WAG 4 contaminants is considered to be unlikely.
Model does not consider biotic decay	May overestimate risk	Biotic decay would tend to reduce contamination over time.
Occupational intake value for inhalation	Slightly overestimates risk	Standard exposure factors for inhalation have the same value for occupational as for residential scenarios although occupational workers. The time of exposure is assumed to be the same in the risk calculations for occupational workers as it is for residents.

Table 6-13. (continued).

Uncertainty factor	Effect of uncertainty	Comments and Assumptions
Use of cancer SFs	May overestimate risk	Nonradionuclide SFs are associated with upper 95th percentile confidence limits and radionuclide SFs are central estimates of cancer incidence per unit intake. They are considered unlikely to underestimate true risk.
Toxicity values are derived primarily from animal studies	May overestimate or underestimate risk	Extrapolation from animal to humans may induce error caused by differences in absorption, pharmacokinetics, target organs, enzymes, and population variability.
Toxicity values are derived primarily from high doses; most exposures are at low doses	May overestimate or underestimate risk	Assumes linearity at low doses. Tends to have conservative exposure assumptions.
Toxicity values and classification of carcinogens	May overestimate or underestimate risk	Not all values represent the same degree of certainty. All are subject to change as new evidence becomes available.
Lack of SFs	May underestimate risk	COPCs without SFs, may or may not be carcinogenic through the oral pathway.
Lack of RfDs	May underestimate risk	COPCs without RfDs may or may not have noncarcinogenic adverse effects.
Risk/HQs are combined across pathways	May overestimate risk	Not all of the COPC inventory will be available for exposure through all applicable exposure pathways.

Table 6-14. Sensitivity analysis exposure parameter values and sources.

Exposure Parameter	Modified Parameter Value	Rationale/Source
Averaging Time (Noncarcinogenic)		
Future resident	7,300 days	Adult, noncarcinogenic, 20 years × 365 days. Exposure factors handbook indicates 77% of residents move prior to 20 years.
Exposure duration		
Future resident	20 years	Exposure factors handbook indicates 77% of residents move prior to 20 years.
Exposure frequency		
Future resident (soil exposure)	305 days/year	Assumes no exposure to soil for 60 days/year during periods of snowpack and/or frozen ground (based on professional judgement)
Modified resident	150 days/year	Assumed exposure frequency (based on professional judgement)
Exposure time		
Future resident	12 hours/day	Exposure factors handbook indicates an average of 730 minutes/day are spent on non-residential activities
Modified resident	0.25 hours/day	Assumed exposure time (based on professional judgement)
Ingestion rate		
Future residential (soil)	60.5 mg/day	Exposure factors handbook provides mean ingestion rate
Future residential (water)	1.36 L/day	Exposure factors handbook provides mean ingestion rate

Applying the more realistic exposure parameter assumptions to the future resident resulted in a reduction in the total risk estimated by 50 to 60 percent (i.e., a risk reduction factor of 0.5 to 0.4). The modified resident assumptions resulted in a reduction in the total risk estimated by 60 to 99 percent (i.e., a risk reduction factor of 0.4 to 0.01). For example, the total excess cancer risk estimate for CFA-04 under the future residential baseline conditions is 4E-05. Application of a risk reduction factor of 0.4 to this estimate would result in a risk of 1.6E-05. For the two cases considered (i.e., the future resident and the modified resident), modifying the resident assumptions from a full-time resident to a part-time resident resulted in a greater reduction in risk than assigning more realistic exposure parameters.

***6.6.5.2* Exposure Point Concentration Sensitivity.** Altering the soil exposure point concentrations for the future residential baseline scenario to reflect average rather than 95% UCL or maximum conditions resulted in a reduction in the total excess cancer risk estimated by 3 percent to 73

percent (i.e., a risk reduction factor of 0.97 to 0.27). For example the total excess risk estimate for CFA-04 under residential baseline conditions is 4E-05. Applying a risk reduction factor of 0.15 to this estimate would result in a risk of 1E-05.

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